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ON CONGENITAL HEART AFFECTIONS, ESPECIALLY IN
RELATION TO THE DIAGNOSIS OF THE VARIOUS
MALFORMATIONS.

THE WIGHTMAN LECTURE FOR 1909.*

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IN a Mongol,† aged 5 months, with a single auricle, and an interval between the auriculo-ventricular bicuspid valve and the top of the interventricular septum, there was a loud systolic murmur. This was best heard at a point midway between the apex-beat (sixth interspace outside the nipple-line) and the junction of the seventh costal cartilage with the sternum. The bruit could just be heard in the neck and also in the back faintly, the left side the louder of the two. There was no thrill. In Willcock's case‡ there was a loud systolic all over the front of the sternum and most intense between the third costal cartilages, and cyanosis was occasionally

* Delivered at the Section for the Study of Disease in Children of the Royal Society of Medicine, June the 24th, 1909.

† 'Reports of the Society for the Study of Disease in Children,' vol. viii, pp. 278 and 339.

‡ 'Path. Soc. Trans.,' 1887, vol. xxxviii, p. 96.

present. In John Thomson's case* the bruit was loudest at the fourth left space close to the sternum, and it was well heard over the left lung behind.

A diastolic bruit as well as a systolic has been described in "pure" cases of this defect.

Combining my clinical experiences of the "pure" defect with those cases of patent septum ventriculorum, where there has been atresia of the pulmonary artery and where the septal deficiency has been the sole murmur-producing spot, I have found the systolic bruit audible all over the chest, back and front. Sometimes the bruit is conducted into the great vessels of the neck, but sometimes not, and it may be heard there at one examination and disappear the next. In my experience, also, there is considerable variation in different cases as to the area of greatest intensity of the systolic bruit. Sometimes the bruit is most striking at the junction of the left sixth and seventh costal cartilages with the sternum, sometimes it is heard best at the fifth left interspace near the sternum, sometimes at the fourth, sometimes at the third, sometimes at the second, and sometimes midway between the apex-beat and the sternum, and but rarely at the apex. The seat of maximum audibility is also apt to change with respiration; thus a bruit which may be best heard at the fifth left interspace with inspiration may become loudest with expiration at the third left interspace. The bruit is apt to be affected by posture also; thus it may be heard loudest at the apex when the patient is erect, and at the base when lying down. In some the bruit is heard loudly all over the cardiac area below the level of the second interspace or third costal cartilages, but not above them. Another disturbing feature is that it is often exceedingly difficult to satisfy oneself as to the area of maximum audibility, for even while the examination is being conducted the murmur seems to vary in relation to this feature. When the bruit is audible over a large area of the chest it is always heard better over the left side than the right.

It is not therefore possible to lay down hard and fast rules as to the area of maximum intensity of the systolic bruit occasioned by patent septum ventriculorum. It is located on the left side of the sternum on the costal cartilages or interspaces, somewhere between the second left interspace and the junction of the seventh costal cartilage with the sternum, sometimes between the nipple and the left margin of the sternum, and rarely at the apex. The systolic thrill which occasionally accompanies it is not confined to any par-

* 'Edinburgh Hospital Reports,' vol. ii, p. 293.

ticular cardiac area; it is sometimes to be felt in the episternal notch, sometimes at the epigastrium. An important feature in the diagnosis of patent septum ventriculorum is the detection at the apex of a healthy first cardiac sound audible through the bruit and the tactile sensation accompanying it of the mitral valvular snap. The second pulmonary sound at the left base is accentuated if the pressure of the right ventricle be increased, and exceeding purity of this sound is important and suggestive.

The oldest patient that I have seen with perforate septum ventriculorum was a man who died suddenly at the age of 41 years while passing a turnstile at Chester races. He was very blue, the ears, eyes, and face being dusky. The fingers were dusky, and there was a slight suspicion of clubbing, but only a suspicion. Ophthalmoscopic examination of the eyes did not indicate venous stasis. The vessels were not tortuous. The veins were large, and only a trifle darker than the arteries, which were dark. The apex beat was in the fifth interspace, a quarter of an inch external to the nipple. There was no obvious extension of dulness to the right. The bruit was systolic and whistling, heard best midway between the nipple and the left sternal margin, and was not conducted far in any direction. The first and second sounds at the apex were normal, and the second pulmonary accentuated. His pulse was slow, of good volume, but with occasional intermissions.

As in defective interauricular septum, so in defective interventricular septum infarcts may be carried from the pulmonary valves through a defective interventricular septum to the arterial system. In a girl of mine, aged 6 years, whose left heart was healthy, the left middle cerebral artery was blocked in this way, and there were infarcts in the spleen and one kidney. In the child aged 8 years previously mentioned, there were vegetations on the right side of the patent septum.

Defects in the Interauricular Septum.

Defects in the interauricular septum.—A patent foramen ovale is so frequently found at autopsy that it can hardly be classed with other congenital abnormalities, and Fisher * states that slight patency of this foramen can be found in one quarter of adult post-mortems. A widely open foramen, however, must be looked upon as an anomaly. It occurs either alone uncommonly, or frequently in association with other interauricular septal defects. Defects in the lower part of the

* 'Reports of the Society for the Study of Disease in Children,' vol. ii, p. 263.

interauricular septum are rare, and are explained by failure of the septum primum to descend and unite with the endocardial cushion between the ventricles. An example of this deformity in an infant, aged 9 months, I showed to the Society last year.* The auricular septum was represented by a translucent membranous partition of semilunar outline hanging from its roof, attached back and front, and with the arc of the bow at the top, and the base of the bow hanging free and falling far short of the interventricular septum. There were associated cardiac anomalies. In a form that is rarer still the defect is situated above. An example of this defect in an infant, aged 6 months, I recently showed to the Section.† There was a round communication between the two chambers about $\frac{1}{4}$ in. in diameter. Behind, the foramen was bounded by a narrow translucent fibrous structure, the extreme posterior part of the defective septum. In front the margin of the rounded aperture was crescentic when viewed from either chamber, but whereas the crescent on the side of the left auricle appeared single, when seen from the side of the right auricle it was found to be double. This was due to the fact that the anterior part of the auricular septum was divided vertically into two parts, each of the divisions commencing with a crescent-shaped margin. A probe passed between the divisions traversed the septum for nearly $\frac{1}{4}$ in., and then appeared in the left auricle at the foramen ovale. The left division of the septum was thin and translucent, whereas that starting in the crescentic segment on the right side, which was $\frac{1}{5}$ in. further forwards, was thick and opaque. The semilunar valve and the foramen ovale were in the forepart of the partition, and although the foramen was not closed it was very efficiently guarded. The defects in this infant would appear to have involved both the septum primum and septum secundum, the divisions of which in the anterior part of the outer auricular wall were clearly indicated. The only other congenital abnormality found was a bifid apex to the heart. Rarer still the rudimentary interauricular septum may be found below, the upper portions being wanting, or, as in a case recorded by Peacock, there was a large, rounded aperture in front of the closed foramen ovale. Finally, the septum may be entirely wanting, as in a Mongol infant, aged 5 months, shown by me to the Society last year.‡ The

* 'Reports of the Society for the Study of Disease in Children,' vol. viii, pp. 231-237 (illustrated).

† 'Proc. Roy. Soc. Med.,' vol. ii, No. 2, p. 36, Section for the Study of Disease in Children.

‡ 'Reports of the Society for the Study of Disease in Children,' vol. viii, pp. 278 and 339 (illustrated).

associated defects were one large bicuspid valve between the common auricle and ventricles and the passage of the aorta over the right bronchus.

Open foramen ovale alone and along with other interauricular septal defects occurs in association with stenosis and atresia of the pulmonary artery, transposition of the great vessels and occlusion of the tricuspid orifice. Defects in the interauricular septum happen nearly as frequently as defects in the interventricular septum. Very occasionally they are the *sole* lesions, and very slightly exceed in number as solitary lesions the similar conditions found at the interventricular partition.

Patency of the foramen ovale and defects in the interauricular septum are often diagnosed and seldom discovered, and for the reason that they do not often give rise to characteristic bruits—indeed, murmurs are but seldom produced by these abnormalities. Holt once found a presystolic murmur localised at the right base, the only lesion found post-mortem being a patent foramen ovale. In a case recorded by Foster, quoted by Koplick,* there was cyanosis with a varying systolic and presystolic murmur at the sternal ends of the third and fourth cartilages. In the case that I have just related, which was a particularly favourable one for observation as the septal deficiency was the sole lesion, there was a systolic bruit heard best over the second left interspace and all over the front of the chest, better heard on the left side than on the right, and inaudible at the back and in the neck vessels. I am in doubt as to whether I timed this murmur correctly, and whether it might not really have been *presystolic* in time and not systolic. Those who have experienced the difficulty of accurately timing murmurs in the rapidly acting hearts of crying babies will appreciate the obstacles that confront one. Harsh systolic murmurs, however, have been described by other observers as well, but it is not at all clear how a bruit coinciding with the systole of the auricles could possibly be systolic. Frequently, as in my case just related, cyanosis is not a symptom, though cyanosis with paroxysmal increase may be a marked feature, as in the case I drew attention to when dealing with cyanosis.† Gross interauricular septal defects may be carried through life without causing any inconvenience or giving rise to any suspicion as to their presence.

Septal defects become of consequence if the auricles act unequally, and if there arises an overflow of blood into the right auricle, which

* 'Diseases of Infancy and Childhood,' Koplick, p. 641.

† *Loc. cit.*, "Morbus Cœruleus."

produces stasis in the systemic veins. A reversal of the flow into the left auricle may bring about the same conditions by throwing extra pressure on the pulmonary artery and so on the right auricle. A point of clinical interest about defects in the interauricular septum, as in the interventricular septum, is that septic and other particles may be carried from the venous system direct to the arteries of the brain and body or from the arteries direct to the lungs.

Stenosis and Atresia of the Pulmonary Region.

Stenosis of the pulmonary region is one of the most common of all the cardiac malformations, and in relation to atresia it occurs nearly three times as frequently. In the large majority of cases of stenosis the interventricular septum is patent. When this septum is closed the foramen ovale is often widely open. Both of these foetal passages may remain open. In rare instances both the foetal passages are closed.

In atresia the interventricular septum is more frequently found closed than in stenosis.

In stenosis the ductus arteriosus is usually closed, being found open, according to Maude Abbott's collection from the literature, in only about one tenth of the cases, but in atresia it is nearly always open. Of thirteen cases, six of stenosis and seven of atresia, I found the duct closed in all the cases of stenosis and patent in all the cases of atresia except one, and in that case the lungs were supplied by a large branch from a left innominate artery, and not by the ductus arteriosus as is usual.* If the ductus be closed in atresia the septum will be found defective, or the septum and foramen ovale will be open, and vary rarely the foramen ovale only. When there is a defective interventricular septum the aorta, which will then be found large, is frequently transposed to the right, and this irregularity occurs more often in atresia than in stenosis. The aorta may arise from both ventricles over the defective septum, or be thrust more to the right and take origin from the right ventricle only.

The stenosis may involve the whole pulmonary region, there being a hypoplasia of the pulmonary artery and its branches, as in a case I exhibited in 1904 in a child, aged $2\frac{1}{2}$ years. This condition was associated with other congenital cardiac abnormalities.†

* 'Congenital Affections of the Heart,' p. 25.

† "A Specimen of Congenital Morbus Cordis," 'Reports of the Society for the Study of Disease in Children,' vol. v, p. 48.

* According to Keith there is associated conus stenosis in 90 per cent. of the cases. In one form, the common variety, the whole infundibulum may be involved and the endocardium thickened. In the other and less common deformity a so-called third ventricle or separate chamber is formed, being separated from the sinus by a perforated muscular partition. In the first variety the deformity, according to Keith, is due to arrest of development of the bulbus as a whole, and in the other the constriction which remains represents the ventricular origin of the bulbus or a persistence of the lower bulbar orifice.

In some cases the stenosis is valvular in character. It may consist of thickened nodular valve-cusps associated with a normal pulmonary artery and normal interventricular septum, as in John Thomson's case,* or the segments may be fused into a cone, or diaphragm, or a barrel-shaped membrane with a rounded or triangular perforation at the apex. When there are two valves only, the orifice will be found slit-like.

In Norman Moore's case,† a boy, aged 3 years, the perforation was no larger than a medium-sized pin. The ventricular septum and foramen ovale were defective. In a case of my own, in a boy, aged $3\frac{1}{2}$ years, the pulmonary valves were thickened and fused into a cone and the pulmonary artery was only the size of a crow-quill. *There were neither patent foramina nor open ductus arteriosus.* There were associated malpositions of the intestines. The cæcum was attached to the fissure for the gall-bladder by an omentum in which the gall-bladder was included. From this point the large intestine ran to the left iliac fossa and then across and behind the small intestine. The sigmoid flexure was in the right iliac fossa, and the rectum lay on the right side. The aortic valves were thick.

In a case reported by Crocker‡ in a boy, aged 2 years, the obstruction at the pulmonary orifice was due to a perforated membrane which showed no evidence of valvular origin. The pulmonary artery was very small and thin-walled. The interventricular septum was patent and there were other congenital cardiac anomalies.

In transposition of the great vessels the pulmonary valves have also been found fused into a cone. In the case of a boy, aged 8 years, reported by Howard Tooth,§ the aorta arose from the infun-

* 'Edinburgh Hospital Report,' vol. ii, p. 294.

† 'Path. Soc. Trans.,' 1885, vol. xxxvi, p. 176.

‡ *Ibid.*, 1879, vol. xxx, p. 275.

§ *Ibid.*, 1884, vol. xxxv, p. 127.

dibulum, and the pulmonary artery from the sinus of the right ventricle. The pulmonary artery was of fair size, and the valves were fused into a cone with a slit-like aperture. The interventricular septum was deficient at the base, and anteriorly to this foramen was a large opening in the septum across which lay obliquely a large fleshy column, dividing it into two smaller foramina. In Crocker's case* in a girl, aged 13 years, the aorta arose from the left ventricle, and just in front of it was a constricted pulmonary artery whose valves were united into a cone. The foramen ovale was patent and the septum ventriculorum incomplete. There were other anomalies.

Illustrating stenosis from congenital defect dissociated from endocarditis is Cautley's case† in a child aged 11 months. The aorta arose from the right ventricle and the pulmonary artery from the left. The orifice of the latter was stenosed owing to a valvular defect. The valve was bicuspid. There were patent septum ventriculorum and patent foramen ovale, and other cardiac irregularities.

In some cases stenosis has evidently originated later in intra-uterine life, after the closure of the septum ventriculorum as in Thomson's case. In the other and the more numerous variety the forms that are met with suggest a developmental origin and a secondary endocarditis infection implanted on a rudimentary pulmonary tract.

In developmental cases the pulmonary artery is usually thin-walled and vein-like. In the other variety the pulmonary artery may be thin-walled also, but it is often either dilated or normal. In stenosis hypertrophy and dilatation of the right auricle and ventricle are common, and if the aorta be deviated to the right these hypertrophies always arise, and this vessel will be found large and thick-walled. The combination of pulmonary stenosis, deviation of the aorta to the right and patent septum ventriculorum is the most usual of all congenital abnormalities.

In *atresia* the obliteration occurs at the conus, or the valve, or the pulmonary artery may be entirely absent as in Peacock and Reid's case.‡ In a case of my own§ in a boy, aged 19 months, there was no trace of a valve, but the artery was patent from the valve seat

* *Ibid.*, 1880, vol. xxxi, p. 92.

† 'Proc. Roy. Soc. Med.,' Section for the Study of Disease in Children, 1909, vol. ii, p. 162.

‡ 'Path. Soc. Trans.,' 1880, vol. xxxi, p. 91.

§ 'Congenital Affections of the Heart,' p. 83.

to the ductus arteriosus and about the size of a piece of whip-cord. The ductus was the size of the common carotid.

If the septum ventriculorum be closed the right ventricle dwarfs and the rest of the heart enlarges. With a patent septum ventriculorum the right side of the heart hypertrophies and dilates and the left side remains small.

Regarding the detection of right-sided hypertrophy of the heart in pulmonary stenosis and atresia a small amount of right-sided enlargement is rather apt to be overlooked if reliance be placed solely on the superficial area of cardiac dulness. Deep percussion should always be resorted to in addition, and it is well if possible to verify the result by X-ray examination. The lung on the right side of the heart covers a moderate enlargement of the right auricle and ventricle and so masks the signs.

Pulmonary stenosis and atresia supply the most typical illustration of morbus cœruleus, and of the two anomalies atresia affords the most pronounced examples of this condition of body.

The bruit of uncomplicated pulmonary stenosis is systolic in time, and is best heard over the second left interspace close to the sternum or on the third costal cartilage. It is usually conducted over the left front of the chest and crosses the sternum, being heard slightly to the right. It is carried up towards the left clavicle. It usually ends before the axillary fold. There appears to be some doubt as to whether the bruit can be heard in the back in uncomplicated cases. It certainly can—I have post-mortem evidence as to that. It can be heard all over the front of the chest and all over the back, the left side more than the right and also in the left axilla. It is not heard in the carotids.

Should pulmonary stenosis be associated with a patent septum ventriculorum as so frequently happens, a systolic bruit will probably be heard in the carotids. If that be heard the suggestion is that pulmonary stenosis is combined with this septal defect, as I believe I was the first to point out some years ago.* It is not necessary for the aorta to arise immediately over the septal defect to conduct a bruit into the neck vessels. Thus, in a girl, aged 15 months, whose pulmonary artery was guarded by fused, thickened bicuspid valves, with a channel which would admit a probe leading to a thin-walled artery which would allow the passage of a blow-pipe $\frac{3}{16}$ in. in diameter and, with the aorta arising from the left ventricle, the bruit was heard remarkably well in the neck vessels. The septal defect was situated under the aortic valves and admitted the tip of the index finger. In

* 'Congenital Affections of the Heart,' 1894, p. 70.

a child aged 19 months, whose pulmonary artery was absent and whose aorta arose over both ventricles, but more from the right side, the bruit produced at the septal defect was heard in the carotids. There can be no question that in the latter case the bruit was not that of a defective pulmonary track, but was manufactured at the septal defect and carried direct to the aorta. I could give several other illustrations of complicated septal defect with bruits in the carotids and subclavians, but those I have related must suffice.

Sometimes two murmurs of different timbre can be distinguished, one with its area of greatest intensity over the pulmonary area, and due to the pulmonary stenosis, the other lower down on the left side of the sternum over one or other of the interspaces there, or over the situations which have been described as likely cutaneous areas for the bruit of patent septum ventriculorum. While infrequently murmurs of different timbre indicate the combined lesions of inter-ventricular septal defect and stenosed pulmonary tract, it more often happens that the bruit heard is due to the associated septal defect and has not been produced by the narrowing of the pulmonary channel.

In stenosis the second pulmonary sound must in large measure depend upon the mobility, resiliency and size of the pulmonary valves. Under ordinary circumstances this sound may be weakened, but if the right ventricle be hypertrophied, or if the pulmonary artery be dilated or enforced by a large patent ductus arteriosus, accentuation of the second pulmonary sound is likely to occur. The second pulmonary sound should be wanting if there be atresia, but in this condition the aortic second sound is usually very loud and well conducted, and the omission may be missed. X-ray photography may assist in determining an enlarged pulmonary artery and a patent ductus arteriosus. The converse may also be seen by this means, a diminished cardiac shadow over the area of the pulmonary artery denoting its absence in atresia, or indicating hypoplasia of this vessel in stenosis.

A murmur which is apt to be overlooked and which occasionally occurs in pulmonary stenosis is a diastolic bruit. Four times I have met with this, viz. in a girl, aged $3\frac{1}{2}$ years; a boy, aged $3\frac{4}{12}$ years; a girl, aged 11 years; and an infant, aged 10 months. In all the children the *systolic* bruit was heard over a wide extent of the chest, and in three cases in the carotids and subclavians and in the infant also, but it was not of the same quality in this last case. In the child aged $3\frac{1}{2}$ years the systolic bruit was most intense at the second left interspace, in the girl aged 11 years over the fourth

left costal cartilage, in the boy over the left third costal cartilage, and in the infant over the third left interspace. In the three eldest children the right heart was enlarged. The diastolic bruit in all the cases was of the character of an aortic diastolic whiff, and in the two eldest was loudest at the second left interspace and conducted down the left side of the sternum to the epigastrium. It fell short of the left clavicle by half an inch. In the two eldest children the diastolic bruit was most distinct over the junction of the sixth and seventh costal cartilages with the sternum on the left. At the second left interspace in the second eldest child there was a double thrill, together with a systolic thrill in the carotids and subclavians, but the latter was not felt over the epigastrium or apex. In the eldest child the systolic thrill was felt all over the cardiac area. In all the cases the second sound over the aortic cartilage was natural, and in all the pulse tracings were normal. In the boy, who was dusky and had clubbed fingers and toes, an occasional diastolic whiff was heard over the left base. In the infant, who was cyanosed, the diastolic murmur was heard over the third left interspace, where there was a to-and-fro bruit, a harsh systolic and a whistling diastolic, the one rapidly following the other. The diastolic bruit was not heard outside the apex-beat and not to the right of the infant. There was a distinct diastolic thrill to the real left of the diastolic bruit. No second valvular sound was audible over the left base. In the three eldest children there was precordial bulging. There was no suspicion of ulcerative endocarditis in any of these children.

Cautley,* in 1901, showed to the Society for the Study of Disease in Children the heart from a girl, aged 15 years, who developed pulmonary regurgitation, secondary to pulmonary stenosis, complicated by acute endocarditis of the valves. A point about this case was that the regurgitant bruit was present for many months and while the patient was in good health. Cautley in his communication refers to an article by Newton Pitt in Allbutt's 'System of Medicine,' and points out that reference is there made to ninety-nine cases verified post mortem, of which more than half were caused by infectious endocarditis, and of these only two were under ten years of age.

The prospect of life varies in these cases according to the defect. In atresia with a closed septum the children die in infancy. If the septum be patent the child may live a few years in place of a few

* "A Case of Pulmonary Regurgitation," 'Reports of the Society for the Study of Disease in Children,' vol. ii, p. 45.

months. In stenosis with a closed septum middle age may be reached, but with a patent septum adult life is the limit.

About one third of the cases of pulmonary stenosis die of tuberculosis of the lungs. Another though much less likely complication is ulcerative endocarditis.

Ductus Arteriosus.

The average diameter of the *ductus arteriosus* post-mortem at birth is stated by Thérémín to be 4·8 mm., and according to Klotz* this measurement is no indication of its functional capacity during life, for he states, on experimental evidence, that the size of this vessel when in action is equal to the capacity of the pulmonary artery itself. The feature of the artery is its rich supply in muscle and comparative deficiency in elastic tissue. Owing to the changes in the circulation at birth, the induction of a gradually decreasing blood-pressure in the ductus and the contractibility of that vessel, obliteration of its channel normally takes place within a month. Contracture, according to Klotz, is most marked at either end; and wrinkling of the lining membrane probably heralds the process. It is some months, however, before the artery is transformed into the ligamentum arteriosum. In the event of atelectasis soon after birth the duct is apt to remain patent, but not necessarily so. As an explanation for patency in some instances it has been suggested that there is a congenital defect in the wall of the duct, an idea which has been created by the frequent discoveries of anomalies elsewhere in the body. In an infant under my care the aortic valves were bicuspid, and the child a Mongol. In another infant, aged 8 months, there was a patent septum ventriculorum. A series of cases by De la Camp† in six brothers and sisters in which the diagnosis of patent ductus arteriosus was made by physical signs and X rays tends to support the suggestion of congenital defect in the duct to account for patent ductus arteriosus in some cases.

Patency of the ductus arteriosus as an isolated lesion is rare, very few cases being recorded in the literature, but as an associate of other congenital abnormalities of the heart it is quite common. Thus, in the absence of, or a rudimentary condition of, the pulmonary artery, the pulmonary radicles in the lungs are mostly supplied by this vessel. In cases also of isthmus stenosis aortæ of the *infantile*

* 'Trans. Assoc. American Physicians,' 1907.

† 'Berl. klin. Woch.,' January the 19th, 1903.

type, which may be associated with transposition of the vessels, the descending aorta is frequently supplied by this vessel. It is sometimes patent in the *adult* type of isthmus stenosis, and it occurs sometimes in transposition of the vessels. While this duct may remain open with absent or rudimentary conditions of the pulmonary artery, it may be closed in cases of pulmonary stenosis, and not uncommonly is closed. Atelectasis is apt to keep the duct open by increasing the blood-pressure in the pulmonary artery and so in the duct, but not necessarily, because the duct is often found closed in infants, where collapse of the lungs is a feature.

The length of the duct at birth is quite trifling, being only some 15 mm., and, according to Gerhardt, in patent ductus arteriosus of long standing the duct is usually shorter and broader than at birth. The majority of the literature referring to patent ductus arteriosus concerns adult findings. A duct of half an inch long, reported by Walsham,* is one of the longest recorded. It usually does not exceed a couple of centimetres. Rarely the aorta and pulmonary artery communicate directly without any vascular connectant.

The patent duct may be cylindrical or conical, with the base of the cone on the aortic side, or globular. The lumen of the vessel in the cases that have come to autopsy has varied from a bristle to that of a finger. Hypertrophy and dilatation of the right ventricle and dilatation of the pulmonary artery commonly take place in chronic cases. In a girl aged 8 months under my care, not published before, the duct admitted a rod of 6 mm., and a patent septum ventriculorum under the aortic valves, which was its sole associate, admitted a rod of 5 mm. The right side of the heart was hypertrophied and dilated. In another infant, aged 3 months,† its length was about two centimetres, and it directly continued the thoracic aorta. The right side of the heart was dwarfed. Exceptionally the left ventricle is hypertrophied as well. Atheroma in the aorta or pulmonary artery in the vicinity of the patent duct are not uncommon, as also the vegetations of malignant endocarditis. Cyanosis, for the most part, is no symptom of patent ductus arteriosus. In my baby aged 8 months the lips were rather lilac-coloured when crying, but in the infant aged 3 months there was no cyanosis, nor was there blueness in my infant aged 9 months. In Simmons' infant,‡ as in mine, cyanosis only appeared on crying. In Bittorf's child

* 'Path. Soc. Trans.,' vol. xxviii, 1876, p. 43.

† 'Proc. Roy. Soc. Med.,' Section for the Study of Disease in Children, vol. ii, No. 6, p. 262.

‡ 'Intercolonial Medical Journal of Australasia,' February the 20th, 1906.

aged 11 years cyanosis was slight but constant. In Carmichael's* child, a girl, aged 3 years, a case of isthmus stenosis, there was cyanosis and polycythæmia, with clubbing. But here, in addition, the mitral valve was so constricted that there were only three minute orifices in it, each not more than $\frac{1}{8\frac{1}{2}}$ in. diameter.

Attacks of dyspnœa, epistaxis, tachycardia, and angina have been recorded by various observers in association with this condition.

The characteristic rumbling murmur accompanying patent ductus has been verified by post-mortem examination on adults, but not, as far as I am aware, on children. This bruit has been likened to a train in a tunnel, and to the mixed sounds in the engine-room of a screw steamer, and it runs right through the cardiac cycle. Gibson† times the murmur as commencing soon after the systole and gradually shading off in the long pause. Combined systolic and diastolic bruits have been recorded by Drasche,‡ and a diastolic bruit by Fagge.§ Systolic bruits have been described by Murray,|| White,¶ Simmons,** and Bittorf. A bruit corresponding to that regarded as characteristic of patent ductus arteriosus occurs when an aortic aneurysm opens into the pulmonary artery or vena cava. Recently a similar harsh and almost continuous murmur has been described by Hobhouse,†† of Brighton, in a child, aged 7 years, in association with a communication between the aorta and the pulmonary artery. This had taken place secondarily to ulcerative endocarditis of the pulmonary valves.

In my case of an infant aged 8 months just related, the systolic bruit which was present was suggestive of a perforate septum ventriculorum origin, rather than one arising from the patent duct. There was, however, a systolic thrill to be felt occasionally at the left base, which might have been due to either defect. In a girl of mine, aged 9 months,‡‡ with verified patent ductus arteriosus there was a systolic bruit, loudest at the third left interspace near the sternum. It was audible all over the chest, back and front, and heard better on the left side than on the right, and was conducted into the carotids. There was no thrill.

* 'Edinburgh Hospital Reports,' vol. ii, p. 298.

† 'Edinburgh Medical Journal,' 1900; 'Med. Press and Circ.,' May the 30th, 1906.

‡ 'Berl. klin. Woch.,' 1898, p. 1195.

§ 'Guy's Hospital Reports,' 1898, p. 1195.

|| 'Path. Soc. Trans.,' 1888, vol. xxxix, p. 67. ¶ *Ibid.*, 1885, vol. xxxvi, p. 182.

** *Loc. cit.*

†† 'Reports of the Society for the Study of Disease in Children,' vol. viii, p. 337.

‡‡ *Ibid.*, vol. viii, p. 231, *et seq.*

In my infant, aged 3 months,* the bruit was systolic, and masked the other cardiac sounds. It was loudest at the third left interspace, and heard in the front and back of the chest, and in the axillæ better on the left side, and better up than down. It was conducted into the carotids, where it was heard loudly; there was no thrill.

The systolic bruit is heard loudest in the second or third left interspace close to the sternum. It may be heard only to a limited extent, as in White's case, some three inches' radius from its point of greatest intensity; or it may be carried all over the chest, back and front, and into the arteries of the neck, as in my verified cases. A thrill is by no means constant. When it occurs it is best felt in the second left interspace, and if the thrill be carried up to the left clavicle in the direction of the pulmonary artery, that is pathognomonic. A. E. Gibson showed, at the Edinburgh meeting of this Section, an excellent example of this feature in the case of a young woman, who also displayed a rumbling bruit, commencing with the systole and running right into an accentuated second pulmonary sound. A thrill may also be felt in the neck, over the arch of the aorta. The pulmonary second sound may be unaltered, but if the blood-pressure be raised in the pulmonary artery by hypertrophy of the right ventricle or by the contribution from the aorta, then an accentuated pulmonary second sound will be heard. A thrill may be felt in the second left interspace in ostial stenosis of the pulmonary artery or in patent septum ventriculorum, as well as in patent ductus arteriosus, so that a thrill in this situation cannot be considered to indicate any special lesion. If the X-ray picture suggests an enlargement of the pulmonary artery, that point is in favour of patent duct. In Arnheim's case† X rays showed an enlarged shadow of the pulmonary artery fitting on to the cardiac shadow cap-like. Hochsinger figures in Pfaundler and Schlossman‡ this cap-like shadow superimposed upon the heart. The internal mammary arteries are also enlarged in the picture, suggesting a collateral circulation, as was also probably the case in Arnheim's patient, both being indicative of coarctation of the aorta. In Bittorf's case, when viewed from the side, the shadow was the size of a walnut, and pulsed in unison with the aorta. In fact there is now accumulating X-ray and clinical evidence, by various observers, confirming the

* 'Proc. Roy. Soc. Med.,' Section for the Study of Disease in Children, vol. ii, p. 163.

† 'Berl. klin. Woch.,' July, 1903, p. 616.

‡ "Chapter on Diseases of the Circulatory System" in Pfaundler and Schlossman, p. 482. (Shaw and La F  tra).

original observation of Gerhard^t as to the presence in these cases of a narrow band of dulness of a finger's breadth or so to the left of the sternum, occupying the third to the second or first costal cartilages and corresponding interspaces. If the second pulmonary sound be loudly accentuated there will probably also be observed visible diastolic pulsation or rebound in the second left interspace at the time of the closure of the pulmonary valves.

Bruits and physical signs cannot be expected to occur in patent ducts of small calibre. In order that a bruit shall be produced it is necessary that there shall be a sufficient space within the walls of the duct and pulmonary artery, in which the conflicting blood-currents shall operate best to produce vibration and sound. There are now a fair number of cases in the literature where patent ductus arteriosus has been diagnosed but where the patients are still alive and where the diagnosis has not been verified. Among these is a case shown by Cecil Williams,* at the Bristol Provincial Meeting of the Society for the Study of Disease in Children. This child, when first seen by him, had a loud and prolonged murmur, of roaring character, continuous throughout the whole of the cardiac cycle, but loudest during systole. It was heard best in the second left interspace. The murmur, after two and a half years, had been replaced by a faint systolic bruit, and the conclusion of the exhibitor was that gradual occlusion of the ductus arteriosus had taken place.

I have narrated my combined clinical and pathological experiences of patent ductus arteriosus, and I will now add my clinical observations in relation to this matter in the shape of the following hitherto unpublished cases which have come under my notice.

Of the 100 cases previously referred to, in a boy, aged 5 years, a girl, aged 18 months, and a boy aged 2½ years, the physical signs were in favour of patent ductus arteriosus, the murmur in each case being systolic. In a boy, aged 12 years (*vide* Fig. 1), under my care, there were presystolic and systolic bruits at the apex, together with a long, rumbling systolic bruit at the base. The morbus cordis had been known two years. The basic bruit was entirely distinct from the bruits at the apex in timbre, and also by reason of a considerable interval of inaudibility between them. The basic bruit was best heard at the second left interspace, and it was conducted up to the left clavicle and but slightly below the second interspace. It was conducted across the sternum slightly to the right and as shown in the photograph, and it was well marked within the area outlined by the broken line. It was heard in the

* 'Reports of the Society for the Study of Disease in Children,' vol. iv, p. 310.

carotids and subclavians better on the left side than the right, and it was audible behind at the root of the left lung. The bruit ran right up to the second pulmonary sound, which was ringing in character. There was an occasional systolic thrill carried up towards the left clavicle. At times its characteristic was that of a valvular snap sensation rather than a tremor, but it became distinctly

FIG. 1.

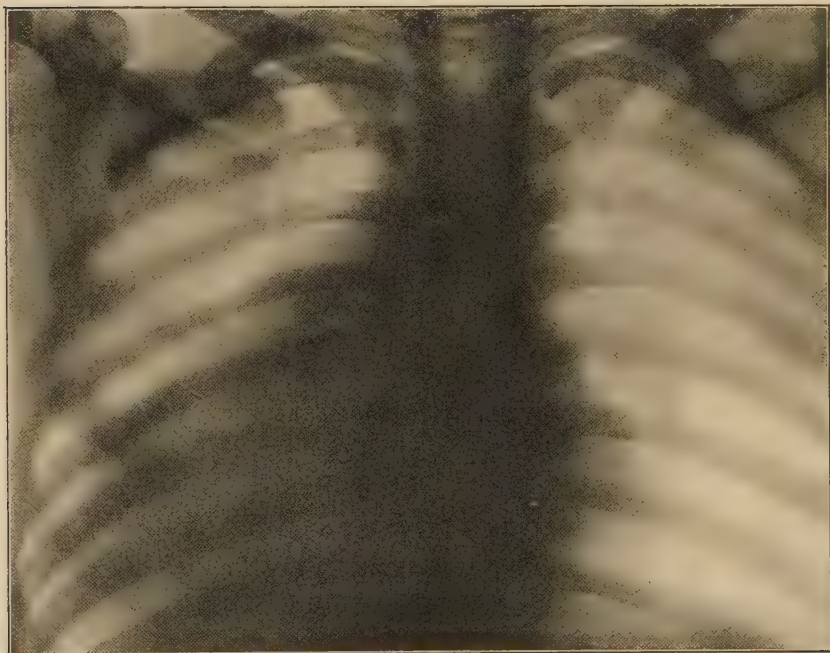


purring on expiration. The second left interspace pulsated visibly with the systole, but not with the diastole. Gerhard's line of dulness was two fingers' breadth in front of the second left interspace. A skiagram (*vide* Fig. 2) showed an enlargement of the pulmonary shadow to the extent of an inch and three quarters in the second left interspace corresponding to the fourth and fifth interspace behind. There was no extension of the cardiac dulness to the right. His mother and three other children were examined

for patent ductus arteriosus, but this was not present in them and they were free from heart lesion.

In a girl, aged 3 years (*vide* Fig. 3), there was a loud, rumbling systolic bruit running right up into the second pulmonary sound, which was accentuated. In addition the rumbling bruit was composed of a peculiar crackling sound like the stringing together of a set of fine pulmonary *râles*. The murmur was best heard in the second left interspace and was conducted in a variety of direc-

FIG. 2.



tions, perhaps a little better up towards the left clavicle than downwards. It was heard but little across the sternum, and did not extend beyond the cardiac area towards the left axilla. It was plainly audible in the carotids and subclavians, better in the left than the right, but it was not heard in the back. There was also a systolic thrill which was felt occasionally in the second left interspace and in the direction of the pulmonary artery to the clavicle. There was no suspicion of cyanosis. Gerhardt's band of dullness was present in the usual situation, a broad finger's breadth to the left of the sternal margin, the upper dotted line in the photograph

being its external limit. The right heart was not enlarged. The child had a mole on its left forearm. A skiagram (*vide* Fig. 4) showed a pronounced dome-shaped structure on the top of the heart opposite part of the first and the second interspaces and third rib in front and occupying the fourth and fifth interspaces behind. The aorta above it was of normal dimensions. The pulsation of this dome-

FIG. 3.



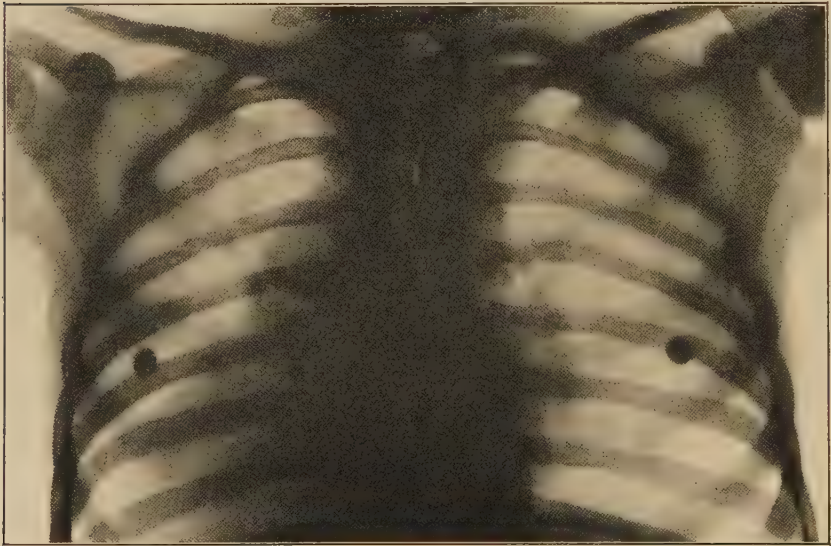
shaped shadow corresponding to the patent duct and enlarged pulmonary artery was synchronous with the cardiac systole. Both parents and two other children were also examined and their hearts were found to be normal.

My last case, a girl, aged 5 years, is of some interest owing to the disappearance of the bruit while under observation. When I first saw her in October, 1894, the systolic bruit, which was heard loudest in the second left interspace, was heard all over the cardiac

area and conducted to the left clavicle. It was audible in the neck vessels. The second pulmonary sound was accentuated. In March, 1895, once or twice I thought I could hear a systolic bruit at the left base, but this was very indefinite. In July, 1896, the basic bruit had completely disappeared, but when she was seen on this occasion she had developed an apical systolic bruit following a rheumatic attack.

Systolic bruits in my experience are the usual accompaniments of patent ductus arteriosus in children, and the rumbling bruit

FIG. 4.



occupying the systole and diastole in adults which is considered to be characteristic of that lesion is not common in them. Williams's case is the only one I have found a record of occurring in a child, and that eventually became systolic.

Tardy closure of the ductus arteriosus may well explain the disappearance of cardiac bruits in children who have been diagnosed as suffering from a congenital heart affection. Reviewing the morbid anatomy of the congenitally deformed heart, closure of this foetal channel appears to be about the only thing possible in the way of a cure open to these cases.

(To be continued.)

IRREDUCIBLE INTUSSUSCEPTION IN THE INFANT TREATED BY ILEO-COLIC ANASTOMOSIS.

By HENRY RUTHERFURD, M.B., F.F.P.S.Glas.,
*Surgeon Glasgow Royal Infirmary; Extra-Surgeon Royal Hospital
for Sick Children.*

CASE.—Norah A—, aged 4 months, was admitted to the Royal Hospital for Sick Children, Glasgow, on March the 7th, 1908, at 1.15 a.m. She had been fed at the breast and was in good condition. The mother stated that on the first of the month the child was fretful, looked pale and vomited occasional mouthfuls, seemed in pain, clenching its hands and drawing up its legs. Next day it seemed well, was quiet and did not vomit, but at midnight began to scream, passed bloody mucus and began to vomit. Vomiting went on throughout March the 2nd and 3rd (being also the second and third days of illness); on the 4th there was some occasional sickness, but on the 5th the child seemed to be very well. On the 6th vomiting again began; at 4 p.m. blood and mucus was noticed on the napkin. On admission the child looked ill, was pale and restless, with sunken eyes. Temperature 99° F.; pulse wonderfully good, about 124. A tumour was easily felt in the abdomen lying across the epigastrium and left hypochondrium, more or less covered by the ribs and cartilages but admitting of being drawn down from under them. Nothing could be felt *per rectum*.

Without any attempts at reduction by indirect methods the abdomen was opened to the left of the umbilicus between 2 and 3 a.m. The intussusception seemed to be of the ileo-cæcal form and its apex had passed beyond the splenic flexure. It was only partially reducible by manipulation; the peritoneum of the intussusciens having begun to crack, the attempt to reduce was given up, and ileo-colic anastomosis was done. The connection was made by a double row of sutures at a point in the transverse colon below the apex of the intussusceptum in its partially reduced condition.

For the first twenty-four hours the child was given merely spoonfuls of water and barley-water. On the morning of March the 8th it was put to the breast, and during the remainder of its stay in hospital was fed by the mother three times a day, her feeding being supplemented during the night mainly by barley-water.

On the day of operation the bowels moved once, on the following day three times, and regularly at about this rate till March the 13th, when there set in some diarrhoea, with green stools: no blood or

sloughy material was seen. The diarrhœa was got over by the 17th. The temperature on the day of operation did not exceed 99° F.; on the next day it rose to 100°; by the fifth day it was normal; at the onset of diarrhœa it rose to 101·8°.

The child was taken home about sixteen days after operation. It was shown to the Glasgow Medico-Chirurgical Society on April the 3rd. It has been seen by me on several occasions since, the last time having been in March, fully a year after the operation, when it was seen to be well grown, plump and in excellent condition.

Irreducible intussusception would seem to admit of treatment by three alternative methods:

- (1) The formation of an artificial anus.
- (2) Resection.
- (3) Exclusion by lateral anastomosis.

Of the first of these I have had no experience, and I do not know of any case in the infant at all events where it has been done with success and the continuity of the bowel re-established by a subsequent operation. In the vast majority of cases the opening will have to be made in the small intestine, and the danger of inanition cannot be left out of consideration, though perhaps it has been too much insisted upon. In cases coming late to operation where the bowel above the obstruction is loaded and distended and more or less paralysed, where every stitch-wound is likely to become a septic sinus, it seems reasonable to think that drainage of the bowel contents is what is wanted and that by the quickest method possible. It is not loss of the intestinal contents that is the impending danger, but retention of them and poisoning from intestinal absorption of them long before any gross extravasation of them can occur by ulceration or gangrene.

In making an artificial anus it would probably be the best procedure to select a loop low down and take it out to the extent of four or five inches through an opening in the flank, tie in a glass tube, and after assisting the adjacent coils to empty themselves, to close the wound of exploration, which is presumably in the middle line.

This, of course, is to be regarded as a temporary expedient. Supposing the child to have recovered, it will, I believe, be best to re-open the abdomen in the middle line and make such a lateral anastomosis as may be practicable between the ileum above the artificial anus and the colon below the intussusception. Such an anastomosis should be free; it is to be for life, and a large stoma will simplify the treatment of the artificial anus. There is no question

of restoring the continuity of the bowel at this point; the ends will simply be freed, cut short down to their intact surfaces, inverted and dropped into the abdomen.

Resection of the parts involved has in the infant been almost uniformly disastrous. The only exceptions known to me are (1) the case recorded by Clubbe, of Sidney, where after effecting reduction by direct manipulation of the intussusception he resected four inches of the bruised and torn ileum and did an end-to-end anastomosis by suture. This was a child, aged 11 months, the only one of nine resections that succeeded.

The other (2) was the case reported by Mr. F. W. Collinson, of Preston. ('Lancet,' October the 17th, 1907). The patient was a girl, aged 3 months. The operation was done seventeen hours after the onset of symptoms. Seven inches of bowel were removed, including a small piece of ileum, the cæcum, and a portion of the ascending colon. The connection was made by a Mayo Robson's button and the button was passed on the fifth day.

I have done resection in the infant for this condition five times and all the cases have died. In two of the cases the method was what may be described as Maunsel's, though that surgeon had not in view resection for existing (pathological) intussusception. It consisted in (1) suturing the entering to the receiving layer, (2) opening the intussusciens, (3) turning out and cutting away the intussusceptum, (4) doing a circular suture of the entering and returning layers, (5) closing the incision in the intussusciens. In the normal bowel, in artificially produced invagination and in diagrams this looks both simple and secure, but the conditions differ from the diagrams. I have seen an entering layer stretched and macerated till it looked like a vein within a returning layer tumefied by œdema and ecchymosis beyond all relation to the contained tube to which an attempt was made to sew it. Possibly the treatment of this difficulty is to see that none of the entering bowel that has been drawn upon and squeezed is used, but that fresh bowel is drawn in and the actual intussusception increased. That may mean increased violence to vessels. At all events the portions of bowel involved are not suitable for use as might be supposed. These remarks have reference to acute intussusception in the infant. The chronic type is widely different as I gather from reports. I have no experience of it. As to acute intussusception in the adult I have only operated on one case; that was not diagnosed beforehand; it was irreducible, and was treated by lateral anastomosis and died. Some years ago I opened a case of acute obstruction in a middle-aged woman and found what I took

for an intussusception in the region of the cæcum. Entero-colic anastomosis relieved her for the time and she went out to return in six months with widely disseminated abdominal cancer.

Pozza ('*Clinica Chirurgica*,' 1901, No. 5; see '*Centralblatt für Chir.*,' 1902, p. 421) recorded a successful case in a woman, aged 37 years. The symptoms were of six days' standing and consisted in abdominal pain, great distension, vomiting, latterly fæcal, and the passage of bloody mucus which appeared on the fourth day; no rise of temperature, no tenderness. Before connecting ileum and transverse colon by a Murphy's button the greatly distended colon was opened and its contents allowed to escape. After the passage of great quantities of fæcal matter, on the seventh day a slough of small intestine 60 cm. long came away. Pozza thought that cæcum and ascending colon were represented.

The patient was dismissed on the eighteenth day and did well subsequently. Pozza lays weight on the prompt relief to the distended bowel and to the rapidity of operating with the Murphy button.

In 1891, Senn, of Chicago, strongly recommended the making of an anastomosis, and practised the operation on animals in which he had previously produced intussusception. He quotes cases in which the same result had been obtained by a process of ulceration resulting in a fistula bimucosa. This can only be regarded as a fortuitous issue in abandoned cases. He says: "Intestinal anastomosis without resection of the intussusception is applicable only in cases of irreducible invagination in which the intussusceptum is only a few inches, at most a foot in length, and in which the external surface of the affected segment shows no indications of the existence of gangrene." On which it may be remarked that a foot of intussusceptum in the infant is a considerable length, and that the intussusciens is not the first part to undergo gangrene.

But Wilms ("Der Ileus," '*Deutsche Chirurgie*') is of opinion that such a procedure is only likely to be attended by a good result when combined with resection at the time or immediately afterwards. It was discussed at the Twenty-second German Surgical Congress, and while Braun recommended anastomosis in special cases, Von Eiselsberg pointed out the dangers (1) from blocking of the stoma by further advance of the intussusceptum, and (2) from inflammatory processes beginning in the tumour.

My colleague, Mr. R. H. Parry, operated with success in June, 1908, at the Royal Hospital for Sick Children. The child, a boy, was aged 6 months. The symptoms were of about forty-eight hours'

duration. The patient has since done well, and Mr. Parry has been unable to detect any surviving tumour. I have to thank him for permission to refer to the case.

I have used the method three times: twice in infants, with one success as detailed; once in an adult man in whom the diagnosis was only made on opening the abdomen after much handling of the parts: he died.

In 'St. Bartholomew's Hospital Reports,' vol. xlv, 1908, Dr. J. L. Maxwell reports four cases of chronic intussusception in the adult treated by lateral anastomosis. The patients were Chinese, all males. Three recovered, one died. The one who died was an elderly and feeble subject; the anastomosis in his case was done by means of a Murphy's button.

Post-mortem there was found no leakage of intestinal contents, but a very small area of necrotic tissue at the under-surface of the button.

In the other three cases which did well the anastomosis was by suture. They were aged 14, 19 and 29 years. Of two cases of excision in the hands of the same surgeon both died.

Dr. Maxwell is able to report that his cases were well and partaking of the ordinary rough feeding of the country (Formosa) three to eighteen months after operation. Mr. Parry's case is well twelve months and my own fifteen months after operation, and these facts seem to justify the opinion that the objections already quoted from Braun and Eiselsberg as to the occurrence of further progress of the intussusceptum and ulceration at the neck or gangrene of the intussusception are not based on any necessary result of the procedure. It is to be remembered that while my own case and Mr. Parry's were in the infant and acute, Dr. Maxwell's were in the adolescent and young adult and *chronic*. But taken together, and in view of the disastrous results of other methods, the cases must be held as presenting a claim for further use of lateral anastomosis to short-circuit the bowel and exclude the intussusception. What becomes of the intussusception we do not know; that it may even be reduced in some cases is possible. Seun says of his experimental cases that the tumour was found to undergo atrophy. Yet it has not in the successful cases recorded given trouble.

We may claim for it that (1) it immediately restores the continuity of the bowel and permits closure of the abdomen; (2) that it is simple and expeditious as compared with any operation of resection, and further, that it may be safer for the patient when complete reduction is only to be accomplished after prolonged manipulation, and where,

though the continuity of the bowel is, indeed, re-established, the parts involved are left bruised, infiltrated and more or less thrombosed and paralysed to transmit the intestinal contents propelled from above, in a condition, that is to say, which may be supposed to facilitate the passage of toxins and micro-organisms into the peritoneal cavity—a process which is attended by the sharp rise of temperature and toxic symptoms so often seen, and with which many operations, apparently successful, end in death within twenty-four to forty-eight hours.

HYPERPYREXIA OCCURRING IN CHILDHOOD.

By JAMES BURNET, M.D.

IF we may judge from the literature of the subject hyperpyrexia appears to be of very rare occurrence in childhood. The two following records of cases in which this was a marked phenomenon will therefore, I trust, prove of interest to members of this Society.

The first was that of a girl, aged 7 years, with a tuberculous family history, the brother having abdominal tuberculosis and both sisters having suffered from tuberculous adenitis. I saw the patient late one evening. The face was greatly flushed, the skin was burning and dry. She seemed only half conscious and swallowing was difficult. The temperature in the axilla was 107° F. There was considerable dyspnoea. Careful examination of chest and abdomen, of throat and ears, etc., failed to reveal the presence of any cause for the hyperpyrexia, and there were no evidences of any infection. Sponging of the chest was first tried, but the temperature only fell half a degree. The child was then wrapped in a blanket and the head held over the front of the bed while cold water was poured over the head from a jug for a couple of minutes. The temperature fell in half an hour to 105° F. Tepid sponging of the chest was resumed and presently the thermometer registered 103·4° F. The child was now less flushed, and was able to swallow a tablespoonful or two of milk diluted with water. Next morning at 9.30 a.m. the temperature was normal, and the child appeared perfectly well.

This case is unique in so far as no cause could ever be ascertained to account for this sudden and great rise of temperature. There was apparently no gastro-intestinal disorder, no pneumonia,

rheumatism, or other disease present. I suspected that there might ensue some infectious condition, but such was not the case. I have since met with a somewhat similar rise of temperature—this time to 106° F. in a highly nervous lady at the onset of what turned out to be a very sharp attack of influenza, but such was certainly not the case in the child whose attack I have just referred to.

My next case was that of a boy, aged $8\frac{1}{2}$ years, suffering from acute rheumatism. I had been in attendance for a fortnight. He was a very nervous child and easily excited. The heart was considerably dilated but no endo- or pericarditis was present. He had been treated by means of ten-grain doses of sodium salicylate and appeared to be doing well. The temperature had been normal for several days, when suddenly he became excited, crying out that there were "beasts in the bed," and nothing would pacify him. When I saw him some two hours later he was very flushed and excited, and, in fact, I had considerable difficulty in taking his temperature, this time in the groin. The thermometer registered 107.2° F. Cold sponges were held over his head for several minutes, and then the head was douched with cold water in the manner adopted in my former case. The temperature almost at once fell to 104° F. and the child was now more composed. I returned about three hours later and found that it had risen again to 105° F. I ordered sponges rung out of iced water to be applied to his head. Next morning I was told that the child had had a fairly good night, but that he was coughing. His temperature was 103.6° F. Examination revealed well-marked pericardial friction. This may have been present on the day previous, as the child's excited state at the time interfered with my examination. This case ran a favourable course throughout, and the patient made an excellent recovery.

In this instance I think we are warranted in assuming that the sudden rise of temperature was due to the development of pericarditis. At the same time I have seen a child who was having large doses of salicylates for a prolonged period become excited, although the temperature did not rise. I am fond of treating psoriasis by the administration of salicylates, and so also chorea, and it was in a child suffering from the latter complaint that I observed an almost maniacal manifestation, which certainly disappeared soon after salicylates were stopped and bromides substituted. There may be some connection here; at all events, it is worth while remembering that salicylates may cause nervous excitement in some children, and even hyperpyrexia, it may be.

Although we must admit that hyperpyrexia is rare in early life,

still I think we are wrong in assuming that it must be less common in the child than it is in the adult. At all events there is evidence to prove that cases do occur, even in the course of rheumatism, although this has been doubted by some writers. It would, indeed, be interesting if our Society were to collect from all the available literature the recorded cases of hyperpyrexia occurring in childhood, and publish the results of their investigations. Meantime, though I need not further refer to the literature of the subject, which must be thoroughly familiar to you all, I cannot but mention the valuable contribution which Dr. George Carpenter has made to it. He has reported three cases of acute nephritis associated with hyperpyrexia and one of chorea.

It appears that renal affections in children are particularly liable to be associated with high temperatures. In pyelitis, for example, we often meet with a temperature of 104° F. and over. Influenza is another disease in which I have occasionally met with a temperature of considerably over 104° F., and it is conceivable that here also hyperpyrexia might occur. Severe vaso-motor disturbances or hysterical fever might also account for it. I have also observed that writers on cerebro-spinal meningitis frequently refer to abnormally high temperatures as occurring in their patients. In pneumonia associated with cerebral symptoms and in enteric fever temperatures of 105° and 106° F. have been placed on record. Beyond this, however, I regret that I have no further personal experiences or suggestions to offer regarding the kind of case in which hyperpyrexia may be expected to occur.

The treatment, after all, is the most important point. Antipyretics are valueless and even dangerous. I am not quite certain as to the safety of applying an ice-bag over the chest in such cases. It may cause a good deal of shock and even collapse. When the skin is dry I think it is best to try tepid sponging in the first instance. If this has no effect then cold should be applied to the head, either by means of a sponge or an ice-bag (if the case is in hospital). Should these measures fail then I am strongly in favour of cold douching of the head and neck. For this purpose the patient is wrapped in a blanket and the head held over a basin or bath. A jug of cold water is then repeatedly emptied over his head and neck. So far as I can discover no untoward effects are likely to follow such a procedure.

One has carefully to keep in mind that children often bear cold badly, and that the application of ice to the chest may induce pneumonia in very young and feeble subjects. Such a risk is altogether

put out of the question if we substitute cold douching of the head and neck in the manner suggested.

Had time permitted I should have liked to refer to the interesting question of the actual cause of the hyperpyrexia in such cases. I am inclined to the view that there is a toxin present which is operating upon the heat-regulating mechanism and putting it out of equilibrium. We cannot at present do more than theorise as to the causation of this condition, and probably one theory is as good as another. I offer you this one for want of a better. I think hyperpyrexia in childhood deserves more attention than it has hitherto received, and I trust that this Society will be encouraged to investigate it still further.

THE DIFFERENCES BETWEEN THE SEXES IN THE DEVELOPMENT OF SPEECH.*

By ERNEST JONES, M.D., M.R.C.P.Lond.,

Demonstrator of Psychiatry, University of Toronto; Psychiatrist to the Toronto General Hospital.

It is universally recognised by those who have worked at the subject that the articulatory capacity develops in girls earlier and better than in boys. No proof, therefore, need here be quoted of this statement, which I have thoroughly confirmed by my own investigations. Up to the present no satisfactory explanation has been given of the fact, most writers confining themselves to the remark that girls are in general more adept and skilful in their movements than boys. This popular opinion has not been established by exact psychological investigations, so that it cannot be accepted as a satisfactory explanation of the greater lingual dexterity of girls. Further, it is certain that girls' speech surpasses that of boys in other respects than in mere muscular dexterity, indicating that some more recondite factor must be at work. Girls show, for instance, a much smaller tendency than boys to suffer from defects in fluency, particularly stammering (*Stottern*), which are now known to originate in complex psychical causes.

It has seemed to me possible that a clue to the problem might be obtained by making a detailed study of the precise respects in which the articulatory capacity of girls most surpasses that of boys. This

* Read before the International Congress of Psychology, Geneva, August the 6th, 1909.

is best done by determining the frequency of the various articulatory defects presented by average children. So far as I can find out from the literature, previous studies of this kind have all suffered from one of the following fallacies. Either they have been based on the study of too few cases, as in the speech clinics of Gutzmann, Neumann, Oltuszewski and others, or else when the frequency of the various defects in large numbers of children has been recorded, as by Mehnert,* Rouma,† Sarbo‡ and others, the observations have been made by untrained school teachers. Observations made in this matter by school teachers are of hardly any scientific value, especially when they are made by a number of different individuals, of different sexes, whose standard of comparison possesses no uniformity.

The present paper is based on the examination of 450 normal school children, and as the observations were all made personally it was possible to employ a uniform standard. Fifty children from each school year were examined, the sexes being equally represented. The details of the method employed and the results obtained have been elsewhere recorded,§ so that I need here quote only a few of the most important conclusions. The inquiry was limited to the consonantal sounds; 227 of these were tested in every case, and marks allotted according to a conventional notation.

One hundred and thirty-two of the sounds tested were better enunciated by the girls, 63 by the boys. We shall first consider the respects in which the girls most excelled, and then those in which the boys did. The former may be divided into two groups: (1) *linguo-dentals*, and (2) *sibilants*; the superiority of the girls was most pronounced in the first of these groups. (1) There are in English two *linguo-dentals*—voiceless and voiced *th*. The girls excelled with nineteen of the sounds containing a *linguo-dental*, the boys with only one. The superiority of the girls was often very marked, the number of defects being in one instance five times as great with the boys as with the girls. (2) Of the sounds containing one of the four *sibilants*, *s*, *z*, *sh*, *zh*, the girls excelled with 65, the boys with only 4. The boys replaced the *s* sibilant by a peculiar lateral sound (*oral sigmatismus*) in 140 instances, the girls in only 3; they replaced *sibilants* in general by it in 223 instances, the girls in only 13.

The relative capacity of the girls was least developed in the case

* Mehnert, 'Über Sprachstörungen,' 1904, S. 18.

† Rouma, 'Internat. Arch. f. Schulhygiene,' Bd. ii, S. 165.

‡ Sarbo, 'Monatsschr. f. Sprachheilk.,' Bd. xi, S. 65.

§ Ernest Jones, 'Internat. Arch. f. Schulhygiene,' Bd. iv, S. 186, and Bd. v, S. 137.

of the posterior linguo-palatals, and was here actually inferior to that of the boys. Of the sounds containing one of the three posterior linguo-palatals, *k*, *g*, *ng*, the girls excelled with 20, while the boys excelled with 21. The greatest difference between the sexes here was found with *ng*, where nearly twice as many defects occurred with the girls as with the boys. Next to this the respect in which the boys most excelled was with explosive linguo-palatals combined with liquids, particularly the sounds *dn*, *kn*, *gl*.

If we now survey these results we note that the sounds in respect to which the girls excelled differ from those in respect to which the boys excelled in one important matter, namely, in that they are sounds more easily taught to deaf children. They are therefore sounds which a child can learn to imitate not only by hearing, but also by the process of lip-reading. The inference thus seems plausible that the development of the articulatory capacity in girls may proceed by the aid of unconscious lip-reading to a greater extent than in boys.

With the children investigated a fairly close correspondence was found* in each sex between the degree of articulatory development and the amount present of nasal obstruction, and, therefore, presumably of deafness. Deafness occurs to about the same extent in the two sexes, so that the effectiveness of it in retarding articulatory development is greater with boys than with girls. This one would attribute to the fact of hearing being with boys the almost exclusive channel of education of the articulatory capacity. With girls, on the other hand, the effect of partial deafness is not so serious, because they can make use of the other educative channel (vision) to a greater extent than can boys.

If this hypothesis is substantiated by more extended investigations then it will become necessary to proceed a step further and inquire into the reason why lip-reading should play a more important part in the case of girls than of boys. There is no corresponding difference in the visual acuity of the two sexes, so that some influence must be at work allowing girls to profit to an unusual extent from their visual perceptions *in the particular respect in question*. In other words, for some reason girls must be able in intercourse with their elders to learn more from the faces of their audience than do boys, probably because they watch them more calmly and with less embarrassment. This is, I think, in accord with every-day observation, for it will be generally conceded that, before the age of mental

* Fifth Report to the Medical Officer, London County Council Education Department, 1908. To be published in detail later.

puberty—that is, before nine or ten—boys on the whole more often show awkwardness, abashment, and shyness in the presence of their elders than do girls. This may well be due to the more frequent and intense feeling of shame, or even guilt, that boys experience as a consequence of the nature of their early sexual emotions.

THE EXISTENCE AND RESULTS OF TREATMENT OF PULMONARY TUBERCULOSIS IN CHILDREN OF SCHOOL AGE.*

By E. G. HAMILTON WILLIAMS, M.D., D.P.H.

THERE seems to be a wide-spread belief that pulmonary tuberculosis is exceedingly rare in children. A less common view appears to be that children do suffer from it, but the disease in them is usually of such an acute and rapidly fatal nature that one does not see children affected by it in schools, *i. e.* that soon after onset the child is so ill that school attendance is out of the question.

With regard to the latter view, I may say at once that I believe it to be partially true of children under five years, and I think that much of the conflicting evidence on the subject we are discussing is due to the fact that, when the word “children” is used, many men at once think of little children, from birth to five years. For example, in Dr. Walter Carr’s paper on post-mortem examinations of “children” dying of tuberculosis, he states that 202 out of 330 were under two years of age. I have been struck by the excess of young children (under three years) admitted at the childrens’ hospitals, from which much of the clinical material hitherto at our disposal has been drawn, and from reports of which many of our theories have been formed. Now we have in the elementary schools fresh clinical material to consider, and we must be chary of applying to it the theories we have deduced from the consideration of material of a different kind. I believe that the frequency of pulmonary tuberculosis increases, year by year, as the child approaches puberty—as the conditions gradually change from those of childhood to those of adult life. Later on I shall offer you definite evidence on this point.

I will first allude briefly to my own experience and then will give the history of certain children’s sanatoria.

* Read before the School Medical Officers’ Association, in London, on April the 13th, 1909.

A few of the cases I have diagnosed have had the diagnosis confirmed by being admitted to the sanatorium at Knightwick, in our county. (This sanatorium has no beds for children, but has, none the less, accepted children in a few cases.)

One of these children happens to live near me, and I frequently see him. Unfortunately, his home is of the poorest description. He returned from Knightwick looking an absolutely different boy, but he has now lapsed, and has developed hæmoptysis; but, nevertheless, I see him constantly out and about. To my knowledge he has had the disease three years, and he now looks no more ill than he did three years ago. He is now over fourteen years and goes to work. Several other cases have been in the sanatorium, and in each case the duration of the illness has now extended over some three years or more. They have not been rapidly fatal.

I have mentioned these particular cases because in them my diagnosis has been confirmed by the selecting committee and by the sanatorium doctor. But I have had the same experience with other school-children and my private patients. Children have been under my care for years suffering from phthisis, and unless the home circumstances are bad I have not found there is much danger of a fatal termination. I consider children more resistant to pulmonary tuberculosis than adults. And by the word "children" must be understood those from five to fourteen years of age. There appears, however, to be a special tendency, especially in the earlier years, to death from tubercular meningitis, but the pulmonary disease is rarely lethal.

I had no idea till very recently that the existence of phthisis in a more or less chronic form among children was receiving recognition by the opening of sanatoria for them. Until recently, sanatoria were not, I believe, available in England for persons under sixteen years of age. Since 1906 two sanatoria for children have been opened; in addition, since 1904 the Metropolitan Asylums Board has had a home specially devoted to pulmonary tuberculosis among children under its care. There is also a sanatorium at Blencathra, in Cumberland, where about 12 per cent. of the admissions have been children.

Thus both the existence of pulmonary tuberculosis in children, and its amenability to treatment, are receiving recognition in widely different parts of England. And I am informed that there is no difficulty in finding cases; the requests for admission far exceed the accommodation.

I recently visited the Millfield Home. This home is near

Worthing, and was opened in 1904 for the treatment of cases of phthisis. Dr. Cecil Last is the medical officer to the home, and was most kind. He showed me cases and gave me figures which are very interesting. From Dr. Last's reports in the 'Annual Reports of the Metropolitan Asylums Board' I find that the number of children admitted during the years 1904-07 was 284. To these must be added the number admitted since then.

These children include every variety of pulmonary tuberculosis, from early to late.

On going through the records, Dr. Last picked out no fewer than thirty-two cases which were sent to him with cavities already formed in their lungs.

Of these thirty-two children, three have died, one is still in the home, and the rest have been discharged. After deducting those three who have died, and those who were over fourteen on admission, I get a list of twenty-five children whose ages vary from three upwards, and whose average age is just over nine years. These children stayed in the home, taking an average, twenty months each. One was in forty-four months, and several over thirty months. These children, while at the home, were up and about, playing with the others, and attending school. No doubt they were at times in bed with acute exacerbations, temperatures, etc., but as a rule they were in fair health.

Thus, it is evident that in spite of advanced pulmonary tuberculosis these children were, most of them, living pretty happily for some twenty months at Millfield. Many of them were discharged on the ground that they were not likely to receive permanent benefit, as the disease was too advanced. And in spite of this, the majority did not look ill. In fact I remarked to Dr. Last that if one had to rely on merely picking these children out of school, and not on systematic examination, one would miss the larger number, and Dr. Last agreed. In short I submit that these cases support the view that pulmonary tuberculosis is very frequently a chronic process in children just as in adults, and that it is not necessarily either acute or rapidly fatal.

To quote Dr. Last's own words from his report for 1904: "Sixty-two cases were admitted and eight discharged, leaving fifty-four children under treatment. Some of these are advanced cases, but the progress they are making is very satisfactory, and their physique, considering the severity of the disease, remarkable. The results that are obtained in other cases are most encouraging; the gain in weight is obvious, and their general appearance of health,

even after a short stay, is a striking contrast to their appearance on admission.

"The cases that have reached us are most instructive, and illustrate the fact that children in early stages of consumption, if placed among suitable surroundings, have great recuperative power, a complete restoration to health being confidently expected in a great number."

Another sanatorium for children, only, exists at Holt, in Norfolk, and I am indebted to Dr. J. B. Gillam, the medical officer, for the following facts: "Of sixty-five cases, thirty were admitted 'late,' and sixteen were admitted when the disease had been in progress for a year or more without making much headway, and so, from a clinical point of view, might still be classed as early. Nineteen cases were admitted early. In the majority of the cases the diagnosis was evidently made some time after the disease had started. This I gather from the reports of doctors signing admission forms. In reply to the question, 'Give duration of disease,' a big '?' is the common reply. The question of diagnosis is a most difficult one. If one is to wait for proof by finding the tubercle bacillus in the sputum, one may wait in many cases till the death of the child. . . . Hardly one in twenty of my children expectorates. I maintain that the clinical symptoms are sufficient for diagnosis."

This is the view held by Sir Clifford Allbutt, who considers that a case that is not diagnosed till physical signs are present is a case bungled.

In addition to Dr. Gillam's letter, I have also his printed reports on the sanatorium for the years 1906-07 and 1907-08. They are especially interesting because the cases are, where possible, followed up and reported on again in the following year.

The cases are divided into early, moderately advanced, and advanced cases.

Of "early" cases there were six in the first year and they were all discharged "apparently cured." These six children are reported on the following year. One was lost trace of, four remained well, and one returned to the sanatorium. This last case is an interesting one, and is reported as follows: *First year*, "K. H—, girl, aged 8 years, remained twenty-six weeks, gained 10 lb., went away apparently cured." *Second year*: K. H— re-admitted. This child went home in January, 1907, in excellent condition, to live in a London basement. In June, 1907, she was re-admitted with a large cavity in one lung and the disease rapidly spreading. She remained eleven months, gained 10½ lb.; the cavity dried up and

she was sent away improved very much. She is to go to a country home."

This case was under observation, therefore, with an interval of six months, from July, 1906, to May, 1908, a period of twenty-two months—certainly a chronic case, though with considerable disease.

Of the cases in Class II, that is, more advanced cases, the following are interesting.

"V. J—, girl, aged 9 years, remained thirty-eight weeks, gained 11 lb. Although considered a doubtfully hopeful case, as both lungs were affected, did exceedingly well, and returned home with disease apparently arrested."

She is reported on a year later: "V. J— has slight cough, otherwise is fairly well; looks, eats, and sleeps well, and is growing very tall. Lives in country."

The next case is somewhat similar. "L. G—, girl, aged 4 years, remained twenty-five weeks, gained 9 lb. Showed improvement, but the disease was still persistent."

A year later the report is: "Keeps well and is able to attend school regularly."

Then comes Class III, advanced cases:

"E. H—, aged 12 years, remained twenty-five weeks, gained $8\frac{1}{2}$ lb. Although improved in general health, was unable to make any headway against the disease, which remained about the same as on admission."

A year later the report is: "Has rather lost ground since her return from Holt."

But, I would observe, even this case remained without much change for a year and a half.

The above children are not cases picked out as being especially resistant to the disease, but are fair average samples of children with phthisis. It will be seen that the fatal cases are proportionately very few, and that by far the majority are what may be termed "chronic" cases.

We may deduce the same conclusion from the report of Dr. Allinson on the Children's Sanatorium at Stannington, Northumberland. The sanatorium was only opened in February, 1908, and he reports on forty-eight cases. Every kind of case was accepted—good, bad, or desperate—and yet he writes as follows: "All of the forty-eight patients hitherto received have done well, except four. Some of our cases were in the last stages of cavity, fever, and emaciation—one from a workhouse with cavities in both lungs making striking progress. From a limited number one cannot draw

conclusions, but I venture to think the treatment of tuberculosis of the lung in children more hopeful than that of adults."

The results of treatment are much the same at Blencathra. Dr. Goodchild, the medical superintendent of the sanatorium, reports as follows: "The total number of children admitted amounts to 12 per cent. of the total admissions of the year. It is during childhood that infection occurs in most cases. With regard to the proportion of children of school age who are affected with tuberculosis much difference of opinion still exists; this is due largely to the paucity of data up to the present, but there seems to be a gradually increasing disposition to place the proportion much higher than was at one time held to be true. Of the 115 patients under treatment during the year (1908), fourteen were children of school age, and many others were seen by the Superintendent, but accommodation could not be provided for them. Most of the children have been going to school, but a few were diagnosed as tubercular on admission to Carlisle Infirmary for other complaints; results of treatment have in most cases been very satisfactory—a better average, class for class, than with adults."

In regard to Blencathra, the complaint is the same as expressed by Dr. Bulstrode in his exhaustive report for the Local Government Board on sanatoria in general—that the cases are not sent early. Dr. Goodchild speaks of the very low percentage of patients presented for admission in the early stages. In 1907, 9·8 per cent. were classed as early, and in 1908 only 4·9 were early. Yet all the others must have been early at some time. In future we will hope that the early stages will be diagnosed by the school doctor.

The evidence of these four sanatoria for children with their 500 odd children shows clearly that children respond extremely well to treatment, and that with it, even in advanced cases, the disease is often arrested. They respond even better than adults, or, in other words, the disease is of a sufficiently chronic nature to allow the time for the necessary processes of repair. From the lay point of view the children would not be considered ill. They are in such a condition that, if at home, they would be attending school, and very often do not even to the medical eye present the appearance of being ill. Many of them look in far better health than other children of the same class without disease, but who happen to live in unfavourable circumstances.

But we know from post-mortem records among adults that many people have had pulmonary tuberculosis and have recovered from it without any special treatment. They resist the disease successfully.

But under treatment children resist the disease better than adults. Is it not also probable that without treatment they resist it at least as well as adults? Or, to express the question differently, Is it not probable that as many children manage to fight the disease successfully while attending school as do adults in their ordinary lives?

I said at the beginning of this paper that I would bring forward some evidence to show that phthisis becomes more frequent as children approach the age of puberty.

Dr. Young writes that as age progresses the protective mechanisms become in general progressively more evolved.

Again, he says that, "Fibro-caseous or chronic pulmonary tuberculosis in children conforms to the characters observed in adults. Rare before six years of age, it becomes more common after that age, and the lesions are less often apical than is the case in adults, but clinically they differ but little from corresponding cases in adults." He further states that the cases are often chronic in course, and of fairly good prognosis under favourable circumstances.

Dr. Young's experience is confirmed by other writers from a different standpoint—that of the death returns.

Dr. R. W. Philip analyses the death-rate from pulmonary tuberculosis among children in Scotland. He shows that the death-rate from phthisis from ten to fourteen is nearly double that from five to nine.

(The children are grouped in four age-groups, namely, under one year, then those of one to four, five to nine, and ten to fourteen. Between 1901 and 1905 the death-rate from phthisis per 10,000 of the population for children under one year was 4·6. From one to four years of age it is 4·4, and from five to nine it drops to 3·2, but from ten to fourteen it rises to 6·1.)

He states that, "The most remarkable, truly astounding fact which seems fairly deducible is the part which the *school life* of the child seems to play in increasing liability to, and mortality from, tuberculosis." Further on he says: "The facts seem to constitute a grave indictment against ordinary school life and urgently call for serious consideration." Dr. Philip concludes his paper by urging the need for special open-air schools for tuberculous children, and also the pressing duty incumbent on school boards to reform the ordinary class-room and to demand much freer ventilation.

Personally, I think so many factors must be considered that I cannot agree with Dr. Philip as to the effect of school life. The fact that the death-rate from phthisis of the girls is so much higher than that of the boys points to some other important factor.

In addition to the demand for better ventilation I would add the need for efficient warming. The Board of Education gives 60° F. as the minimum temperature permissible in the Infants' Department of a school. It is a common thing to find the temperature below 50° in these and other departments, and teachers have not seldom informed me that in cold weather the temperature has been below freezing-point when the school opened at nine o'clock, and very little better three hours later. The ordinary open grate by itself is utterly inefficient for warming large class-rooms, and it is criminal to keep children, especially infants, in class at such temperatures. When seen in these surroundings they are blue with cold, and the windows are all—very naturally—shut. To keep children for hours in cold, stuffy class-rooms is the worst possible treatment for any cases of early phthisis which may be present.

The point, raised by Dr. Philip, is of such interest that I obtained from the Registrar-General the corresponding figures for England and Wales. These figures show the same thing for England as described in regard to Scotland and Ireland.

The annual mortality from phthisis per million in England and Wales for both sexes in the ten years 1891 to 1900 was as follows: "From five to ten years of age, 206; from ten to fifteen, 368." For the five years 1901 to 1905 the same thing is shown, as follows: "From five to ten years of age, 172; from ten to fifteen, 287." In other words, the very high mortality from phthisis between the ages fifteen to thirty-five, a mortality that accounts for one third of all the deaths occurring at those ages, has really set in before the age of fifteen. Instead of considering phthisis as a disease demanding recognition from fifteen years onwards, we must realise that it has become an important factor some five years earlier—that is, from the age of ten or thereabouts.

The Registrar-General's figures show clearly that the steady increase in phthisis mortality has set in by the age of ten, instead of at fifteen as seems usually believed.

Hence, in order to treat phthisis successfully, one must sort out the cases in school so that one may undertake the cure at an age when we have the most chance of success.

Abstracts from Current Literature.

Medicine.

Two cases of congenital hemi-spasm of the lower lip in infants ('*La Clin. Infant.*, January 1, 1909, No. 1, p. 1).—**G. Variot** and **M. Bonniot** reported these to the Soc. des Hôp.; both were boys. Case 1 was aged 12 days; when he cried the mouth was awry like cases where the facial nerve has been pressed on by forceps, but close examination showed that the orbicularis palpebrarum and other muscles were normal. As soon as the child ceased crying the grimace disappeared and the face became normal, although the lower lip seemed slightly inclined to the right and the buccal slit was not absolutely horizontal. The head was always turned to the right when lying in the cradle. When six months old the deformity still was apparent on crying but in a less degree, and also on laughing there was slight twisting of the mouth, but to a less degree than on crying. In the middle of the neck a transverse oblique on the right side was noted as if the platysma on this side contracted more than on the other, where no fold existed. Case 2, a month old, presented an exactly similar aspect except that the twisting was on the left side. On palpating the two halves of the lower lip in repose, it was noticed in both infants that there was no appreciable difference in consistence. The electrical reaction of all the facial muscles was normal except half the orbicularis of the lower lip, and indicated a kind of *atony* limited to a single muscle, the result of which was to produce a kind of spasmodic grimace when certain muscles came into play. The electro-diagnostic examination of this muscle was interesting: it reacted normally to faradism but to galvanism there was a partial reaction of degeneration, and indicated an arrest of development of the muscle. In order to explain the peculiar aspect of the lower lip when these infants cried, it must be borne in mind that the orbicularis oris forms a sphincter on which the elevators and depressors of the commissure are fixed, *i. e.* for the lower lip the *triangularis*, the *quadratus*, and the *platysma*. These three muscles in the cases before us, not finding sufficient counter-resistance, retract abnormally on the lower lip of the same side, producing the spasmodic grimace.

VINCENT DICKINSON.

Gastric radioscopy: vomiting in an aërophagic infant ('*La Clin. Infant.*, January 1, 1909, p. 13).—**A. Lesage**, in a paper read before the Soc. de Biologie, says that the normal nursling is aërophagic, and that when this aërophagy becomes excessive gaseous distension of the stomach provokes vomiting. It is well known that in the adult vomiting may depend on the same mechanical cause. The air swallowed by the nursling during a feed issues from the stomach in inverse proportion to the milk which enters the gastric cavity. When the air swallowed is in excessive quantity, the stomach is filled with a small quantity of milk and a large quantity of air; the resulting distension provokes a violent contraction and expulsion of air and milk. The fact of vomiting being produced in this way has been demonstrated by gastric radioscopy. It is enough in such cases in order to prevent vomiting to watch the feeds, giving plenty at longer intervals. Sometimes aërophagy is accompanied by spasm of the cardia; the air swallowed cannot escape from the stomach when once it has got there, and will only be expelled when the distension attains to such a degree that the stomach repels; then vomiting

ensues owing to violent gastric contractions. In a second group of cases, *acrophagy* with spasm of the cardia, as shown by radioscopy, the feeds which are not followed by vomiting are those in which only a small proportion of air has entered, due to small feeds given at short intervals. Radioscopy thus shows us the existence of a variety of vomiting in the nursing not hitherto described—the vomiting caused by excessive *acrophagy* with or without spasm of the cardia. It is a fact that the vomiting of inanition and of spasmodic gastritis often depend on one or other of these varieties.

VINCENT DICKINSON.

Observations on the leucocytosis produced by the toxin of the diphtheria bacillus, with especial reference to the changes which follow the injection of antitoxin (*Journ. of Path. and Bact.*, vol. XII, p. 154).—**H. R. Dean** draws the following conclusions from experiment performed in the Physiological Laboratory and in the Jenner Clinical Laboratory at St. Thomas's Hospital. (1) After the experimental injection of diphtheria antitoxin there is great leucocytosis, due to an increase in the number of polymorphonuclear cells. (2) After the injection of the diphtheria toxin degenerative changes can be demonstrated in both the white and red cells. In the latter polychromatophilia is often well shown. A reduction in the number or a complete disappearance of the coarsely granular eosinophilic cells is, as a rule, observed. (3) No myelocytes were seen. (4) The changes in the blood which follow the experimental injection of diphtheria toxin are similar to those seen in cases of diphtheria occurring in the human subject. (5) A dose of antitoxin given simultaneously with the toxin completely protects the animal and prevents leucocytosis. (6) When a sufficient dose of antitoxin is administered within a short time of the injection of toxin the number of leucocytes rapidly falls to normal. (7) Where a considerable interval elapses between the injection of toxin and the injection of antitoxin no reduction in the leucocytosis occurs. (8) In two cases of diphtheria in children the interval between the onset of the disease and the employment of antitoxin was relatively short, and in these cases a considerable reduction in the leucocytosis appeared to follow the use of antitoxin. (9) No reduction in the leucocytosis was observed in four cases in which there was a relatively longer time before antitoxin was used. (10) A marked reduction in the leucocytosis within the first twenty-four hours after the injection of antitoxin probably indicates a good prognosis. (11) A sufficient dose of antitoxin completely protects the blood-cells from degenerative changes.

JAMES E. H. SAWYER (Birmingham).

Juvenile tabes and general paralysis from acquired syphilis (*Arch. de Med. des Enfants*, July, 1908).—**E. Apert, Lévy-Fraenkel, and Ménard** report the case of a girl, aged 15 years, suffering from tabes which terminated with general paralysis. At the age of 2½ years she contracted syphilis from her parents. Her father died from tabes and general paralysis. The mother contracted syphilis from her husband, and later developed signs of tabes. The patient had the following symptoms: Romberg's sign; eyes—inequality of pupils, Argyll-Robertson's pupil, slight irido-choroiditis; sensation normal; knee, Achillis tendon, and triceps-jerks absent; speech normal; fibrillary tremor of tongue; mentally, very obstinate and liable to fits of temper, slight hallucinations, no grandiose ideas. The authors quote six other cases: (1) General paralytic infant, mother having tabes and

general paralysis (Gianelli). (2) Child, aged 13 years, general paralysis; father general paralytic, mother tabetic (Kutner). (3) Child, aged 15 years, tabes; father tabetic (Brusch). (4) Tabetic child; father tabetic (Babinski). (5) Child, aged 15 years, tabes; father died of general paralysis (Linser). (6) Child, aged 15 years, tabes; mother tabetic (Cantonnet).

JAMES E. H. SAWYER (Birmingham).

The cutaneous and ophthalmic tuberculin test in infants (*Arch. of Pediat.*, 1908, p. 801).—**H. L. K. Shaw** employed these tests on eighty-one infants under twelve months of age in whom there were no clinical and physical signs of tuberculosis. None showed any reaction to the ophthalmic test, in which both human and bovine tuberculin of various strengths had been employed. The cutaneous test gave one positive reaction. This was in an old case of empyema with a discharging sinus. The pus was injected into a guinea-pig, which died later of tuberculosis. Shaw concludes that these tests are not reliable in infants, since less than 1·5 per cent. react to either test, whereas the percentage of tuberculosis in infants ranges from 2·3 to 19·9 per cent.

J. D. ROLLESTON.

Empyema and purpura (*Arch. of Pediat.*, 1908, p. 838).—**F. Huber**.—A boy, aged 2½ years, with no history of tubercle, syphilis, or any hæmorrhagic tendency, was admitted to hospital with the signs of empyema. Numerous petechiæ were present on the limbs and buttocks, relatively few on the trunk, and there was a small ecchymotic area round the exploratory puncture. There was no swelling of the joints nor periosteal hæmorrhage. Operation was delayed for a few days, and orange juice and gelatine were given to improve the blood condition. On incising the pleura a large mass of clotted blood followed by a considerable quantity of pus was evacuated. Complete recovery took place.

J. D. ROLLESTON.

Hæmorrhagic nephritis in mumps (*Pædiatrics*, 1908, p. 627).—**J. E. Hunt**.—A girl, aged 5 years, a few days after a mild attack of mumps became drowsy and feverish. The urine was scanty and contained much blood. Generalised œdema developed; complete recovery finally resulted.

J. D. ROLLESTON.

Typhoid fever in infants (*Pædiatrics*, 1908, p. 663).—**J. L. Morse** records four cases in children whose ages were 11, 16, 18, and 19 months. All were admitted within one month to the Infants' Hospital at Boston, in which there had been only two other cases within the last twenty years. Widal's test was positive in all on admission. All showed rose spots, and in three the spleen was palpable. In three the pulmonary signs were unusually prominent. The duration of the fever was comparatively short. There were no marked nervous symptoms.

J. D. ROLLESTON.

The doctrine of difficult dentition (*Pædiatrics*, 1908, p. 595).—**T. J. Elterich** in twenty years of pediatric practice has never seen a single case which could be properly diagnosed as difficult dentition. Acute otitis media, which may exist without any symptoms referable to the ear, should always be suspected in cases of pyrexia without obvious cause.

J. D. ROLLESTON.

Pediatric study in London and Vienna (*Pædiatrics*, 1908, p. 681).—**H. M. McClanahan** thinks that Vienna excels in opportunities for laboratory

work and private instruction, and London in its enormous amount of clinical material.

J. D. ROLLESTON.

Pneumonia occurring simultaneously in brother and sister (*Gaz. des Hôp.*, 1908, p. 1779).—**Railliet**.—A boy, aged $4\frac{1}{2}$ years, and his little sister, whose age is not stated, were attacked at eleven hours' interval by pneumonia. Both had been sleeping in the same bed. A second brother, aged 2 years, who had been sleeping in a separate room, escaped. The sister had a mild attack; the brother's illness was more severe. Pain in the region of the umbilicus and right iliac fossa, with vomiting and absence of auscultatory signs in the chest was the cause of the boy being admitted to a surgical ward as a case of appendicitis. It was not till the third day that the diagnosis of pneumonia was made. The greater severity of the brother's attack is probably to be attributed to diminished resistance in consequence of repeated attacks of appendicitis, mumps, and a recent sore throat.

J. D. ROLLESTON.

Lymphangioma of the orbit (*Med. Press*, March 10, 1909).—**Bergmeister** exhibited at the Gesellschaft der Aertze of Vienna a child, aged $2\frac{1}{2}$ years, with a thick upper eyelid on the left side, which was due apparently to an ill-defined soft swelling extending under the skin to the temporal region. The swelling was somewhat compressible, but was not affected by compression of the jugular. The ball of the eye was not protruding, but, on the contrary, was sunken in the socket. The fundus of both eyes was perfectly normal. In addition there were numerous pigmented patches over the chest and back. According to Recklinghausen's theory this is the first symptom of neuro-fibromatosis in the cutaneous nerves. This neuro-fibroma, sometimes known as Ranken's neuroma, is difficult to distinguish clinically from lymphangioma, as both depend upon the same anatomical process.

T. R. WHIPHAM.

Neuro-fibromatosis (*Med. Press*, March 3, 1909).—At the same Society **Zumbush** presented a man, aged 22 years, with a large, dark, hairy, pigmented mark on the left cheek and temple. Other thick, dark, bristly patches were scattered over the trunk and arms, ranging from the size of poppy-seeds to the palm of the hand. They were sharply defined and the skin around was unchanged. In the patches were concentric rings, rising in tiers, of a livid colour and hard, but some of them were soft and could be pressed level with the healthy skin. The right tibia from the middle outwards had a sabre appearance, while posteriorly it was crooked and irregular. The foot was in a condition of pes planus valgus. The fibula under the Röntgen rays appeared as a thin bristle, but not bent or irregular like the tibia. All the changes were considered to be congenital morbid productions.

T. R. WHIPHAM.

Erythema infectiosum (*Med. Press*, March 3, 1909).—**Escherich** also showed two children with what is known as erythema infectiosum, which, he considered, would be more correctly described as erythema multiforme. The children had a rash resembling scarlet fever and measles combined. It is epidemic and attacks children of from four to twelve years of age. The youngest patient he has ever seen suffering from the disease was fourteen months. The rash appears on the face without any constitutional prodroma in the form of bluish-red, slightly raised patches extending

from the *alæ nasi* to the ears, the forehead to a great extent escaping. The lymphatics below the ears are usually tender and painful to the touch. Along the extensor surfaces of the limbs, as well as in the gluteal region, the morbiliform rash is met with in large patches, but it is less pronounced and not so well defined over the trunk. It usually lasts from eight to fourteen days with varying intensity, having a reticulated marble appearance before disappearing. Lymphatic enlargement, except below the ears, mucous catarrh and general disturbance are all absent, neither are there any sequelæ or other complications. It is evident that during an epidemic scarlet fever, measles or *rötheln* may be easily confounded with this disease. Some authors, like Hebra, draw distinctions between this infectious disease and erythema multiforme, although both agree in the localisation and efflorescence of the rash, but differ in respect to contagion, affirming that erythema multiforme is not infectious, while erythema infectiosum is. Escherich expressed some doubt about the contagious nature of the disease, as he had not found it to spread in hospital, where he had frequently met with it. He thought that both these morbid changes might be included under the term "erythema multiforme." Ehrmann agreed with Escherich that this was only an abortive form of erythema multiforme exudativum, and was not an acute exanthema.

T. R. WHIPHAM.

Diphtheritic paralyses (les paralysies diphtériques) (*Gaz. des Hôp.*, January 18, 1908, pp. 75 and 111).—**Chéné** gives a very full review of the subject. The article does not lend itself to abstracting. A good bibliography is appended.

ERNEST JONES (Toronto).

Congenital pyloric spasm and congenital hypertrophic stenosis of the pylorus in infancy (*Amer. Journ. of the Med. Sciences*, July, 1908, vol. cxxxvi, p. 1).—**Henry Koplik** gives a general account of the condition and reports fifteen cases, each of which is separately discussed. He holds that the term covers a number of different pathological conditions, having different prognoses and treatment. He is strongly in favour of medical treatment.

ERNEST JONES (Toronto).

Peculiarities of the symptomatology of rheumatism in children (*Amer. Journ. of the Med. Sciences*, July, 1908, vol. cxxxvi, p. 66).—**C. H. Dunn** calls special attention to the frequency with which rheumatism in children may make its onset with cardiac symptoms alone. He says that it begins with arthritic symptoms in only 40 per cent. of all cases. There are seven clinical types of rheumatism in children: (1) The mild arthritic type; (2) the severe arthritic type, occurring in older children; (3) latent type, beginning with fever; (4) mild primary endocarditis; (5) severe primary endocarditis, characterised by fever and cardiac incompetency; (6) mild pericarditis; (7) severe pericarditis, always with effusion.

ERNEST JONES (Toronto).

Pathology, prophylaxis, and treatment of the umbilical cord (*La Semana Med.*, September 24, 1908).—**Chueco** after observing that one chapter of medicine, that of tetanus and erysipelas of the new-born, will soon pass into history, gives an account of infective diseases of the umbilicus. He recommends that the bath-water for new-born infants be previously boiled and then cooled to the proper temperature, and that the bath be sterilised by heating with alcohol, and, as most important, that the nurse

should wash her hands thoroughly each time before dressing the navel, and small lesion be attended to carefully and thoroughly. M. D. EDER.

Two cases of mumps in sucklings (*St. Petersburg med. Wochens.*, November 22, 1908).—**Hollmann's** first case was an infant, aged 9 months. Many of the family had suffered from mumps, including the mother. The right parotid gland was affected in this infant; beyond slight rise of temperature there was nothing specially to be noted. The second case was a girl, aged 11 months; there had been other cases in the house. When the child was brought there was suppuration in the left parotid gland, which was incised. M. D. EDER.

Total paralysis of seventh right facial nerve in a boy, aged 7 months (*Allg. Wien. med. Zeitung.*, November 24, 1908).—**Hochsinger** showed this case at the Pediatric Section of the Vienna Medical Union. Forceps was employed at birth, but no paralysis was then noticed, and the child thrived splendidly till it was six and a half months old, when it was unwell for a few days, the doctor who attended finding no cause. On October 2 it was apparently quite well; on the morning of the 3rd the paralysis was found and the child was feverish. A fortnight later the paralysis remained. There was no disease of eye or ear, and the diagnosis of peripheral facial paralysis was confirmed by the reaction of degeneration. The author, after dealing with other possibilities, believed that it was a case of elective disease of the nerve related with the then prevalent epidemic of acute anterior poliomyelitis. M. D. EDER.

A new skin phenomenon in sucklings (*Wien. klin. Rundschau*, September 13 and 20, 1908).—**Blattner** has for some time been investigating this reflex, which was first observed by Pfaundler, who noticed, in the case of an extremely atrophic infant, that whenever the skin of the abdomen was touched the lower limb of the same side was drawn up. Pfaundler also observed, in similar cases, that when the relaxed abdominal skin was gently touched there appeared a peculiar wrinkling and puckering of the skin of the lower limb. This spread rapidly over the whole surface, back and front, and after the skin had remained a little while in this condition, disappeared. Pfaundler called this the "shagreen skin-sign." At first sight it seems to be identical with the goose-skin reflex, but Blattner shows that there is only a certain resemblance. Incidentally he remarks that this latter sign has never been thoroughly examined; the only good account of it is to be found in Mackenzie's article in *Brain* (1893). The shagreen skin-reflex was obtained by gentle stroking, rubbing, or massage of the abdomen. The reflex usually occurred upon the same side, but in some cases both lower limbs reacted. The reflex started in the groin, spreading upwards, and then the whole skin was affected down to the ankle, the extensor more markedly than the flexor surfaces. No reflex occurred in the upper limbs, abdomen, chest, or back. It was sometimes obtained when the upper or lower part of the thigh was stroked, but in this case was more marked when the abdomen was stimulated. In a few cases it was produced when the napkin was taken off or by blowing upon the skin. The reflex was not obtained by using ice, hot or cold water, or by faradic current. The sign is very evanescent, but can be reproduced again and again. It only occurs in badly nourished children with relaxed skins, whose cushion of fat has largely disappeared. As soon as the children improve with better nourishment and there is a renewed

turgescence of the skin the reflex cannot be obtained. In healthy sucklings and in all who are more than one year, whatever their atrophic condition may be, there is no reflex. Blattner, therefore, does not regard this as a physiological sign like goose-skin, and he gives reasons for regarding it as a true reflex brought on by contractions of the muscle in the upper layers of the skin. Further observations may lead this reflex to be regarded as of some diagnostic value in the diseases of sucklings.

M. D. EDER.

Pathology.

Splenic anæmia in children (*Munich Society for Children's Diseases*, March 5, 1909).—By splenic anæmia **Benjamin** understands a disease of early life which sets in at the end of the period of suckling, and in which is developed an anæmia of a varying degree with decided enlargement of the spleen. The disease may cease in the second or third year of life, or the child may succumb in that period to the primary disease or some complication. The blood-count in individual cases shows great variations: a severe form of the disease may occur with a normal blood-count, and slight cases with decided diminution of the erythrocytes. The colour index is normal, or only slightly below normal; the leucocytes vary from 5000 to over 100,000; the myelocytes may be enormously increased or be entirely absent, while normoblasts and megaloblasts are always met with. The disease is always accompanied by more or less severe rickets. As in rickets a reduction of the polynuclear neutrophiles is met with; splenic anæmia, however, shows itself essentially in the predominance of non-granular cells of bone-marrow. An exhaustion of bone-marrow may be considered to be the commencement of the disease, indicating a high degree of myelopathy. As compensatory processes are extra-medullary foci of blood formation whereby erythropoiesis and indirectly leucopoiesis occurs. Uncomplicated cases must always occur, with a relative reduction of polynuclears. As the disease shows a constant increase in the large mononuclears, the author considers whether these cells are not the cause of the splenic tumour in children, and whether they do not take a share in the myeloid metaplasia of this organ in the splenic anæmia of infants.

J. E. BULLOCK.

The Spirochæte pallida and congenital syphilis (*Journ. of Path. and Bact.*, January, 1909).—**McIntosh** has studied the distribution of the *Spirochæte pallida* in congenital syphilis. It is found in greatest numbers in the liver, and it is also present in the lungs, spleen, suprarenal glands, kidney, and skin. In the skin, as in the liver, it is in enormous numbers. The author failed to find it in the placenta or in the umbilical cord. He suggests, therefore, that the infection is maternal, the spirochætes being carried in the blood to the liver, where they multiply, and that thence they are distributed to the rest of the body. The organism has a special affinity for structures of the nature of connective tissue or glandular epithelium, and it is especially plentiful in the connective tissue around the blood-vessels.

T. R. WHIPHAM.

Polyarteritis acuta nodosa and periarteritis nodosa (*Journal of Path. and Bact.*, vol. XII, p. 31).—**W. E. Carnegie Dickson** in a paper on the above subject describes the case of a boy, aged $14\frac{1}{2}$ years, who was admitted into the Royal Infirmary, Edinburgh, in May, 1906. The case was thought to be probably one of tuberculous or possibly pneumococcal

meningitis. At the post-mortem examination the following conditions were found: Nodules of polyarteritis acuta nodosa on the small and medium-sized arteries of the heart, liver, spleen, kidneys, mesentery, brain, and spinal cord; subacute nephritis and infarcts in the kidneys; rupture of, and hæmorrhage from, aneurysmal dilatations upon surface vessels of brain; peculiar infiltration of lungs. Evidence of old-standing tuberculosis of mesenteric and other glands. Microscopically the earliest discoverable changes are found in the outer coat. The process rapidly spreads inwards so that the most complete destructive lesions are to be found in the muscular coat. These are accompanied by local inflammatory changes, and are followed by giving way of the internal elastic lamina and the other coats of the vessel wall. Thrombosis of the contents of the lumen or aneurysmal dilatation occurs with proliferation changes in the outer and inner coats of the vessel. Secondary changes are—infarction, necrosis, hæmorrhage, etc., in the organ or tissue supplied by the affected artery. The infection reaches the vessel wall possibly through the vasa vasorum, or perhaps by the perivascular lymphatics. The cause is almost certainly some bacterial or other infective organism or its toxin. Staining for all ordinary pathogenic bacteria and for the *Spirochæte pallida* gave negative results. The condition has to be distinguished from periarteritis nodosa, which is a true periarteritis, nodular in distribution, and in the majority, if not in all, of the cases is syphilitic in origin. The author quotes from literature adult cases and also the following cases in children of polyarteritis acuta nodosa: Female, aged 10 years (Eppinger); infant, aged 2½ months (Krzyszowski); boy, aged 14 years (Jansó and Veszprémi); male, aged 18 years (Mönckeborg).

JAMES E. H. SAWYER (Birmingham).

Surgery.

The operative treatment of pericarditis ('*Wien. klin. Rundschau*,' November 8, 1908).—**Venus**, after giving the indications for paracentesis, regards the presence of pus, which has been certified by exploratory puncture, as necessitating pericardiotomy after previous resection of a rib. This should be followed by careful irrigation with sterilised salt solution and drainage. The prognosis of any pericarditis treated by operation depends almost entirely on the original cause of the disease.

M. D. EDER.

Atresia recti ('*Med. Press*,' March 3, 1909).—**Moszkowicz**, at the Gesellschaft der Aertze of Vienna, showed an infant, aged 1 month, whom he had operated upon for atresia of the rectum. He first made an incision over the left iliac fossa and opened the sigmoid flexure, whence a large quantity of meconium escaped without any subsequent peritonitis. After the child had recovered and the bowel had been fairly emptied an attempt was made to complete the opening at the rectum on the seventeenth day. Before commencing the operation the *cul-de-sac* was washed out and a fluid solution of metallic bismuth was poured in through the fistula and the part examined by the Röntgen rays. It was then seen that the *cul-de-sac* passed down close to the perinæum. A pair of forceps was inserted and the part distended, so that a circular opening could be made from the outside to form an anus. The mucous membrane of the bowel was then brought down and stitched to the skin. The child has since been able to pass its motions by the natural channel, while the fistula closed without any other interference. There was, however, subsequently a tendency to narrowing of the lumen of the bowel.

T. R. WHIPHAM.

Myogenic contraction (*Med. Press,* February 24, 1909).—**Erben** also exhibited a child, aged 3 years, who, having recovered from a broken arm, had all the symptoms of myogenic contraction. During the treatment the fingers assumed a claw-like appearance under the bandages. There was no lesion of the nerves, and the phalanges after extension contracted firmly, showing that the conducting power of the radial nerve was intact. There was no muscular atrophy in the hand or fingers, as usually occurs in peripheral paralysis. He thought that the cause was either a fibrous inflammatory retraction or possibly an ischæmic condition of the muscles. Moszkowicz was of opinion that the contraction was due to ischæmia, which was not infrequent in fractures of the lower third of the humerus. This lesion was usually attributed to a rupture of the cubital artery producing a hæmatoma and so preventing the formation of a collateral circulation. In this way the whole group of muscles of the forearm is affected. It is possible that a necrosis and degeneration of the fibrous tissue may be induced, but it is purely of a myogenic nature and never under any circumstances akin to neurogenic contraction.

T. R. WHIPHAM.

Stricture of the œsophagus; with a report of eight cases from the Children's Hospital of Philadelphia (*Univ. of Penna. Med. Bull.,* December, 1908, p. 298).—**A. P. C. Ashhurst** reports eight cases of œsophageal stricture in children below the age of seven; three were only two years old. The cause was in seven cases the contraction of an ulcer following burns from lye. In most of the cases the stricture had fully developed in under a year. The distance from the dental margin varied from nine to twenty-three centimetres. The various operative procedures are fully considered.

ERNEST JONES (Toronto).

An appendix abscess in a twenty-seven months child; with an analysis of infantile appendicitis in the Johns Hopkins Hospital (*Bull. of the Johns Hopkins Hosp.,* February, 1909, p. 31).—**T. W. Churchman**.—Notes of nine cases of appendicitis in children under five years of age are here given. They were all operated upon and seven recovered; the two deaths were due to general peritonitis. A very interesting review of the subject based on previous publications and on the Johns Hopkins records is then added. From the latter the following facts may be quoted: Of 1223 cases of appendicitis admitted to the surgical clinic 9 were under five years of age and 199 under fifteen (16 per cent. of the whole series). Of 1001 cases recorded by McCosh 17 (1·7 per cent.) were below the age of five, and 153 (15·3 per cent.) under the age of fifteen. The diagnosis is discussed and the following rules laid down: "All urinary symptoms in children should suggest the possibility of appendicitis, and in infants with apparent hip-joint disease, particularly if the thigh be flexed, the same possibility should be kept in mind. Cathartics should never be given for constipation unless it is certain that appendicitis is absent. Palpable resistance on the right side by rectal examination is one of the most frequent findings; the importance, therefore, of rectal examination in any doubtful case is obvious. Pathologically, two features of the complaint differentiate it from the adult cases: (1) a tendency to early perforation, and (2) the frequency of early peritonitis.

ERNEST JONES (Toronto).